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ANEURYSMS OF THE DURA MATER*

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My purpose in this report is to discuss briefly the clinical manifestations of aneurysms of the dura mater. Two such cases were observed; in one a diagnosis was possible during life. In case 1 there was clinically no idea that the lesion of the brain could have been brought about by an aneurysm.

REPORT OF CASES

CASE 1.—*History.*—A hospital clerk, aged 27, was admitted to the ear, nose and throat department of Saint Stephen's Hospital on March 8, 1927, complaining of a "running nose" and fever. Later, severe headaches followed. On March 17, the patient was operated on for a deviated septum. On March 23, he became unconscious and was transferred to the neurologic department.

Examination.—At the time of transfer a right-sided paresis of the musculature of the lip associated with continuous twitchings was noted. The right arm was paretic. Anisocoria, sluggish reaction of the pupils, well pronounced rigidity of the neck and priapism were also noted. The lower extremities were hypertonic; Babinski, Kernig and Brudzinsky signs were negative. There was paralysis of the urinary bladder. The patient was extremely restless and continually touched his forehead with his hand.

The heart was apparently normal; the pulse rate was 80, and the beats were rhythmic; the temperature was 37 C. (98.6 F.). The spinal fluid showed xanthochromia.

The clinical symptoms were considered to be due to meningitis originating from the nose.

Course.—On March 24, the patient's condition became worse. Respirations were of the Cheyne-Stokes type; all the extremities were hypertonic; the pupils were mydriatic and fixed; incontinence of urine and stools was present; the deep reflexes were absent. The immediate cause of death was respiratory failure. The diagnosis was meningitis, probably due to direct perforation of the sphenoidal sinus.

Necropsy.—The postmortem examination (Professor Baló) revealed an aneurysm, the size of a bean, situated on the left side of the tentorium cerebelli. This aneurysm had resulted from an embolism. Rupture of the aneurysm had caused a meningeal hemorrhage. An ulcerative endocarditis was found on the aortic valves and on the aortic leaflet of the mitral valve. The aneurysm was situated on the left side of the tentorium at about its middle portion behind the petrous bone and covered by clots. Coagulated blood also covered the anterior and middle scala as well as the posterior part of the tentorium and falx. The clots extended down to the clivus. The perforation in the aneurysm was the size of a

* Submitted for publication, August, 1928.

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poppy seed. The gyri of the left hemisphere were very much flattened. Histologically, the clots presented the signs of organization. The source of the bleeding was histologically proved to be an aneurysm filled in part by fibrin.

Comment.—During life no signs referable to the heart were noted; aneurysm due to embolism could therefore not have been considered as a possible diagnosis.

Case 2.—This patient, a girl, aged 19, observed some time later in consultation with Professor Hasenfeld, had suffered from heart disease since the age of 7. An attack of influenza caused decompensation. On March 29, 1927, the patient began to suffer from severe headaches and subsequently developed a left hemiplegia.

Examination after the attack revealed a positive Babinski sign on the left side. On the right side there was a positive Kernig sign. The right lower extremity was flexed at the hip and knee joints. Both disks were choked.

Comment.—As a result of earlier investigations on pachymeningitis hemorrhagica, it was concluded that the combined signs which affected both lower extremities could be caused by a right-sided lesion of the brain. This process could be responsible for the left hemiplegia. The contraction of the right lower extremity with a positive Kernig sign could be explained by the increased pressure existing also in the left hemisphere. The presence of bilateral choked disks was also in keeping with such a localization.

I reasoned in the following manner: An intracerebral hemorrhage was not probable because the patient was not unconscious following the hemiplegia. An intracerebral hemiplegia, embolic in origin, could also be excluded since bilateral signs were present. I therefore concluded that the symptoms were due to an extracerebral process. Having considered the observations in case 1 and since the patient in case 2 had an endocarditis, I concluded that an aneurysm of the dura mater, which had resulted from an embolus, had ruptured.

It was presumed that the bleeding was situated extracerebrally, that the spinal fluid would be free from blood and pleocytosis, and further that the resorption of the extravasated blood would take place.

These suppositions have been proved to be true. At an examination, the spinal fluid was normal; the patient made a rapid recovery. She was soon able to walk and left the hospital on June 3. Soon after her discharge she was readmitted to the hospital with high fever and died on July 1. The cause of death was endocarditis.

Necropsy.—The postmortem report (Professor Baló) verified the clinical diagnosis. The heart showed chronic ulcerative mitral endocarditis with stenosis and insufficiency. The case was to be considered an endocarditis lenta. Associated conditions were hyperplastic splenitis and hemorrhagic glomerulonephritis.

A depression was found on the convexity of the right side of the brain where the anterior central gyrus and sylvian fissure join. A nodule the size of a pea, which corresponded to the depression, was found on the internal surface of the dura. This nodule adhered to the dura only by means of a thin pedicle. The whole internal surface of the right hemisphere showed signs of previous bleeding in the form of brown pigmentation. The same alteration was found on the internal surface of the dura extending down to the middle scala. The surface of the left hemisphere showed no such changes. The nodule described proved histologically to be an aneurysm of the arteria meningea media. The wall of the artery could be recognized only in a part of its circumference. At one point the elastic fibers and smooth muscle fibers were missing and the wall was formed by organized fibrin.

The two cases described behaved differently clinically because of the anatomic localization of the aneurysm. On the basis of these observations, I have attempted to group the signs and symptoms of ruptured dural aneurysms in the following manner: The site of bleeding will naturally determine the clinical picture. If the aneurysm of the dura is situated in the posterior cranial scala, after rupture of the aneurysm the blood will flow forward along the basis of the cranium and backward toward the foramen magnum. In this way it may produce meningeal symptoms which may be combined with signs of paralysis of the basal cranial nerves. Sudden onset of severe cerebral symptoms, severe headaches, rigidity of the neck, bloody spinal fluid, xanthochromia and pleocytosis would all be in keeping with the picture.

On the contrary, following the rupture of an aneurysm situated on the dura above the convexity of the brain, the typical symptoms of hemorrhagic pachymeningitis would develop. This has been reported on in a previous communication.[1] The hematoma in this case covered the surface of the brain, especially the frontoparietal region. The blood can penetrate into the anterior and middle scala, but still the hematoma remains above the tentorium. The clinical symptoms may be the following, varying with the anatomic peculiarities mentioned: (*a*) tumor or pressure symptoms, choked disks, headaches and hemiplegia with spastic reflexes on the side opposite to the lesion; (*b*) symptoms of irritation in the extremities of the same side, convulsions, carphology, state of contraction of the lower extremity without a Babinski sign but with a well pronounced Kernig sign; (*c*) clear spinal fluid and no pleocytosis.

COMMENT

The following comments serve to explain the symptoms. The extravasated blood which covers the internal surface of the dura decreases the space between the dura and bone and is followed by signs of tumor. Of these, the hemiplegia is attributed to the impression of the central region, so it is a focal symptom. This pressure inhibits the function of the pyramidal tract and is followed by hemiplegia and a positive Babinski sign. Parallel with the increased pressure exercised on one side runs the elevation of pressure on the other side, but to a lesser degree. Twitchings in the face, arm, carphology, contraction of the lower extremity and a positive Kernig sign, as well as the observations in the spinal fluid, are extremely important.

Since the extravasation of blood is subdural but extrapial, no blood pigment reaches the subarachnoid space, therefore the spinal fluid remains clear.

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1. Von Sarbó, Arthur: Deutsche Ztschr. f. Nervenh. **92**:216, 1926.

In publishing the report of these two cases of aneurysm of the dura, a rare condition, my intention was to separate this group of diseases from the more common aneurysms in the brain itself. It seems that if recognized early enough, aneurysms of the dura could be made a subject for surgical treatment. A characteristic feature of aneurysms of the brain is that the signs of onset after rupture are gradual. Therefore, it is possible that early operative intervention, when indicated, may save life.

The symptoms described can also be caused by aneurysms which are not situated on the dura. The following table proves that the symptoms vary according as the hematoma is at the base or on the convexity of the brain.

Symptoms Following the Rupture of Aneurysms

Aneurysm at the base of the Brain	Aneurysm on the Convexity of the Brain
Onset: Sudden, then gradual course.	Onset: Sudden, then gradual course.
Signs: Severe headaches; severe rigidity of the neck; often paralysis of basal cranial nerves, especially of those of the posterior cranial scala; bloody spinal fluid; xanthochromia; pleocytosis.	Signs: Severe headaches; little rigidity of the neck; crossed alterations in the extremities; on the opposite side to the focus hemiplegia with a Babinski sign, on the side of the focus symptoms of irritation, contractions and a Kernig symptom; possibly paralysis of basal brain nerves with the exception of those of the posterior cranial fossa; spinal fluid clear, without signs of inflammation; usually markedly choked disks, more pronounced on the side of the hematoma.

MULTIPLE SCLEROSIS

THE SENSATION OF AN ELECTRICAL DISCHARGE AS AN EARLY SYMPTOM *

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At a meeting of the New York Neurological Society held on Oct. 14, 1927, Dr. Wechsler [1] presented a case of particular interest.[2] The patient, a man, aged 24, presented two exceptional symptoms: (1) a peculiar sensation of an electrical discharge that radiated down the vertebral column into the lower extremities, and (2) myotonia, which appeared when the patient was asked to carry out a rapid movement with his left arm. A short time after the appearance of the sensation of electrical discharge, the symptoms of multiple sclerosis developed rapidly. These were: diplopia, spasticity, exaggeration of the deep reflexes, Babinski's sign, clonus, absence of abdominal reflexes, nystagmus and tremor. The results of the laboratory examination were negative. There was mild pallor of the left optic disk.

Dr. Wechsler's communication aroused a discussion in which Dr. Henry A. Riley, Dr. Louis Aronson and Dr. S. P. Goodhart took part. This discussion apparently led to the conclusion that the patient had an organic disease of the nervous system, though certain abnormal symptoms that he presented were considered psychopathic and perhaps of hysterical nature. My comments will be limited to the sensation of electrical discharge.

In a previous communication, Dr. Bollack, my assistant Dr. Nicolas and I [3] described in great detail the phenomenon to which Dr. Wechsler drew attention. The first patient presenting this symptom whom we had the opportunity of observing expressed himself thus: "When I

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* Submitted for publication, Oct. 25, 1928.

* Translated by Walter M. Kraus, M.D., New York.

1. Wechsler, I. S.: A Case of Multiple Sclerosis with Unusual Symptoms, Arch. Neurol. & Psychiat. **19**:364 (Feb.) 1928.

2. Translator's Note: This case was one that had been previously reported by Lhermitte, Levy and Nicolas (Presse méd. **35**:610 [May] 1927). The patient on coming to this country was referred to the translator by Mlle. Levy. He was then sent to Montefiore Hospital, where he was seen by Dr. Wechsler, who became interested and subsequently reported the case (Arch. Neurol. & Psychiat. **19**:364 [Feb.] 1928).

3. Lhermitte, J.; Bollack, and Nicolas, M.: Les douleurs à type de décharge électrique consecutive à la flexion céphalique dans la sclérose en plaques, Rev. neurol. **2**:56 (July) 1924.

lower my head I feel a violent shock in the neck and a pain resembling that produced by an electrical current which travels through the entire body from head to foot, following the vertebral column."

Just as in the case of the patient described by Dr. Wechsler, a complete picture of multiple sclerosis appeared gradually after this early manifestation.

After our first article, Dr. Nicolas and I, with Dr. Gabrielle Levy,[4] took up the study of this question again and added the reports of two new cases in which a sensation of electrical current and discharges appeared in the same way as in the first case. Besides a shock that resembled, as the patient told us, electrical discharges, we described sensations in multiple sclerosis which were closely allied to this but which were different in certain particulars. These sensations were not those of shock but of an electrical current, which the patient said he felt going through him.

This sensation of an electrical current was spontaneously produced and appeared periodically. One of our patients wrote: "This resembles a little the ringing of a telephone bell. For example, three seconds of ringing, four seconds of silence and so on during a period which varies from a few seconds to twenty minutes."

It is known that disorders of sensation, that is, dysesthesias, are among the most frequent phenomena occurring in the initial stages of multiple sclerosis. In this instance, however, a specific sensation occurs that is not a pain. The phenomenon that my co-workers and I described is characteristic of the disease. The patients are never deceived as to its character once they have felt it. These sensations do not often appear when the patients are completely at rest, but are almost a continuous accompaniment of those ordinary activities of life that cause flexion of the spine and particularly of the cervical part of the spine. The strange, unexpected sensation of an electrical discharge is disconcerting; it disturbs the patient and is frequently followed by a feeling of anxiety and apprehension. As I have said, this peculiar sensory discharge marks the beginning of multiple sclerosis, and it is only some time after its disappearance that the patient manifests objective symptoms that make a diagnosis certain. What has been learned about this curious sensation has made it possible to diagnose multiple sclerosis very early.

There is only one known condition of the spinal cord that may be a cause of this type of sensory discharge, and that is concussion. In this condition, I described, at the same time that Babinski and Dubois

4. Lhermitte, J.; Levy, G., and Nicolas, M.: Les sensations de décharge électrique symptôme précoce de la sclérose en plaques, Presse méd. **35**:610 (May) 1927.

did, pains having the character of electrical discharges, and the thesis of Ribeton, written at my suggestion, contained many case reports that demonstrated the phenomenon. There is not any need, however, of entering further into a discussion of the occurrence of these symptoms in spinal concussion, for a differential diagnosis between concussion and multiple sclerosis is rarely difficult. In the aforementioned article, I indicated the pathways supposed to be affected when the sensation of electrical discharge is felt, and proposed a hypothesis, stating that I believed that these pathways that are responsible for the sensation of electrical discharge are analogous to those responsible for formication produced by trauma of peripheral nerves.

To return to the case of Dr. Wechsler—there is no question but that in this case the sensation was one characteristic of multiple sclerosis, and that the sensation corresponds to that which Bollack, Nicolas and I [3] had previously described.

The patient presented by Dr. Wechsler was, it must be admitted, an electrician; that his profession did not play any part in the comparisons that he made of his sensory disorders with electrical discharges I do not doubt. The same perhaps does not apply to the other phenomenon to which my American colleague has alluded, namely, the illumination of a lamp by contact of hands, independently of an electric circuit. As Dr. Riley justly remarked, it is not the metallic filament contained in the bulb that illuminates, but simply the external surface of the bulb rubbed by the hands. It is known that some persons on account of peculiar secretions of the skin, can produce electrical sparks by rubbing the skin.

I may add that recently Mondeil [5] succeeded in rendering electric bulbs luminous by rubbing them with the hand. Under the influence of this rubbing, the metallic filaments of certain bulbs contract and thus become curved. This author has put himself to a good deal of unnecessary trouble to demonstrate that in this illumination it is not a question either of fluorescence or of the action of some vital fluid, since it is only an ordinary electrical phenomenon. Whatever the cause of the illumination of the electric bulb may be, however, it has little interest here, since I am considering only the sensation of electrical discharge.

The frequency of the sensation of electrical discharge must be rather high, for since our last article a number of neurologists have observed patients with this sensation, for example, Trioumphoff [6] and

5. Mondeil, G.: Études 20, February, 1928.

6. Trioumphoff, A.: Symptôme de décharge électrique de la sclérose en plaques, Presse méd. **35**:948 (July) 1927.

Roger, Reboul-Lachaux and Aymes.[7] In all of their patients, the sensations were almost of the same character as those that Bollack, Nicolas and I [4] described.

The present article has been written less on account of the apparent strangeness of the symptom described than because of its great significance in pointing the way to a diagnosis of multiple sclerosis at a period when objective signs are rare and the diagnosis is doubtful.

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7. Roger, H.; Reboul-Lachaux, J., and Aymes, G.: Dysesthésies rachidiennes à type de décharge électrique par flexion de la tête dans la sclérose en plaques, Rev. neurol. **1**:1052 (June) 1927.

THE ARGYLL ROBERTSON SIGN IN MESENCEPHALIC TUMORS *

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AND
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SAN FRANCISCO

Cases of mesencephalic tumor associated with Argyll Robertson pupils in nonsyphilitic patients have been reported several times, but it does not appear to be generally recognized that this combination is of definite clinical and localizing significance.

In a recent book entitled "Modern Problems in Neurology" one of us (S. A. K. W.) [1] devoted a chapter to the Argyll Robertson sign, and there recorded three personally observed instances of the combination, in only one of which, however, was the diagnosis confirmed by pathologic examination. With Rudolf, the same writer reported a characteristic example,[2] in which the tumor had destroyed precisely the anterior corpora quadrigemina. At the International Neurological Conference held in Paris, in July, 1928, a similar case, with postmortem confirmation, was described by Professor Guillain.

Other instances from the sparse literature on the subject are that of Moeli [3] (1887), in which a typical bilateral Argyll Robertson sign resulted from a tumor of the third ventricle in a man, aged 57, whose vision was unaffected, that of Farquhar Buzzard,[4] and that reported by one of us (S. A. K. W.[5]), in 1906, in which a colloid tumor of the third ventricle was associated with a clear approximation to the Argyll Robertson phenomenon.

It appears to us appropriate, in view of their relative rarity, to record two further instances of the combination of the pupil sign with tumors of the anterior colliculi, in each of which autopsy has substantiated the topographic diagnosis. In respect to the occurrence of the sign, entirely apart from syphilis, such cases are always important.

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* Submitted for publication, Oct. 12, 1928.

1. Kinnier Wilson, S. A.: Modern Problems in Neurology, London, E. Arnold, 1928, p. 332.

2. Kinnier Wilson, S. A., and Rudolf, G.: J. Neurol. & Psychopath. **3**:140, 1922.

3. Moeli: Arch. f. Psychiat. **18**:1, 1887.

4. Buzzard, Farquhar: Brit. M. J. **2**:1319, 1910.

5. Kinnier Wilson, S. A.: Brain **29**:524, 1906.

REPORT OF CASES

CASE 1.—*History.*—L. T., a woman, aged 17, was admitted to the National Hospital, Queen Square, London, on March 7, 1923, under the care of Dr. Kinnier Wilson, and died on May, 1, 1923. The diagnosis was: tumor of the mesencephalon.

About the beginning of November, 1922, the patient began to suffer every morning from headaches in the forehead and back of the neck, more especially on the left side. The pain was stabbing and lasted usually about three minutes. It occurred every quarter of an hour, roughly, till about 10 a. m. and then did not come on again until evening, often not until the next morning. About the third day of headache she vomited and after the sickness the headache seemed to be worse. The vomiting was not associated with food; she felt it coming and was prepared—sometimes nausea was present, sometimes not. The headaches continued, sometimes accompanied by vomiting, for about three weeks, at which time she found she was unsteady on her feet when she was out for a walk. This unsteadiness continued to become worse until the time of admission. It was accompanied with the sensation of objects moving round the patient from left to right, and as far as she can remember she seemed to go round with the objects, but of this she was not certain.

One day an attack of this giddiness came over her and she felt as though the road was coming up to hit her on the face. She fell, striking her forehead, which stunned her but she was not unconscious. About the same time as the patient began to stagger about she noticed that she saw double, and she had had diplopia at intervals since that time. Singing noises had been heard in the left ear since the beginning of the illness in November. Accompanying the staggering gait was the gradual onset of weakness in the legs and arms, especially in the left leg and left arm.

During the six weeks preceding admission, the eyesight had been failing. All the symptoms described had continued and had gradually become worse until the time of admission. She described the headache as a feeling as if some one were "twisting her up inside her head," sometimes "stirring up the contents of her head."

The menstrual periods had stopped with the onset of the illness, but previously had been fairly regular.

Since the patient went to school, when aged 5, there had been a little tremor in the left arm; this had continued unchanged, till three years before admission, when it gradually became worse and had continued to do so until she was admitted to the hospital.

Examination on Admission.—As the patient sat up in bed, the head was constantly tilted to the left and the occiput slightly approximated to the left shoulder. She constantly closed the right eye when looking at anything. Memory and intelligence were average. Amenorrhea had been present since November.

Cranial Nerves: The sense of smell was normal. Visual acuity was: right, 6/24 +; left, 6/24. The fields were slightly diminished, but this could be accounted for by the papilledema. The fundi showed double papilledema of + 6 diopters. The pupils were widely dilated, circular, central and regular. There was no reaction to light, direct or consensual; reaction on accommodation was prompt. The ocular movements were limited and poorly sustained when the patient looked to the left. When she returned from left lateral deviation to the midpoint there was a slight lack of conjugate movement. The right lateral deviation was not quite full, except with difficulty, but was better than to the left. Upward and downward,

deviation was normal. There was slight internal strabismus of the right eye, and ptosis of the right eyelid. Nystagmus was present on lateral deviation to the right horizontal, about 120 per minute, with the quick phase to the right, slightly irregular; groups of the movements were fairly regular, and moderately fine in type. To the left, after careful testing, the nystagmus was found to be slightly coarser than to the right. There were a few irregular nystagmoid jerks on upward deviation. Downward movements were normal.

Pinprick and cotton wool sensitiveness were diminished slightly on the left side of the face. The masseters and temporals were slightly weaker on the left side, the jaw deviating slightly to the left. The corneal reflexes were present on both sides.

The movements of the upper part of the face were good and equal. The mouth was round in smiling, the movement of the right side being decidedly stronger than that of the left. When the patient showed her teeth the difference was not so marked, but the right was greater than the left. In smiling, the left side of the mouth, instead of moving out, made some twitching movements.

The Weber sign was not lateralized. With the Rinne test, air conduction was greater than bone conduction on both sides. With the Schwabach test, the right was better than the left. The hearing on the right was better than on the left.

The palate was drawn up slightly to the right. There was no dysphagia. The sternocleidomastoids and trapezii were fairly powerful, there being no difference between the two sides. The tongue deviated slightly to the left; there was no atrophy, but a slight unsteadiness was present.

Sensory System: A pinprick was appreciated everywhere, but was less sharp down the left side than on the right. The same was true with cotton wool. The sense of passive movement was normal. The deep muscle sense was slightly diminished in the left arm and leg. The vibration sense was felt slightly less down the left than the right side. Stereognosis was normal. The sense of position in space was normal.

Motor System: The movements of the arms were full and of fair power, the right greater than the left. The power in the trunk muscles was fairly good; there was no deviation of the umbilicus. The movements of the legs were full and fairly powerful, the right greater than the left.

Tone was normal in the right arm and leg; in the left arm and leg, tone was diminished. With outstretched arms there was a marked lowering of the left arm from the horizontal position in a few seconds. When the patient supported the elbows and allowed the hands to dangle forward at the wrist it was seen that there was practically no difference on either side. In the "whip" test, the difference in tone could be made out, though it was slight; also in the sudden knocking apart of the upper arms; with the arms outstretched, again tone was found a little less in the left arm than in the right. In the legs, the left calf muscles felt more flabby than those of the right.

Dysdiadokokinesis: Alternate movements of the fingers and wrists were fairly well performed with the right hand but were slower with the left and with much associated movement of the whole arm. In alternate movements of the toes and feet, again the right was fairly well done but the left was more awkward, slower, and with associated movement of the foot when the toes were moved and of the leg when the foot was doing the alternative movements.

Coordination: The finger-to-nose test was done with a little hesitancy by the right hand, but otherwise was fairly normal. On the left, the nose was not touched accurately and the movement was done in the typical cerebellar winding fashion.

On reaching the nose, the finger could not be kept there, but jerked all over the face, on one occasion, even into her left eye. The heel-to-knee test was done accurately on the right, but with slight unsteadiness when the heel was on the knee. With the left leg, the knee was reached fairly accurately but with a great amount of unsteadiness, and on reaching its position the heel "wobbled" about as the left finger did on the nose.

Bárány Test: With the finger in front, there was no deviation with either hand; but to either side, especially to the left, there was marked deviation of both hands to the left—right hand 2 inches (5 cm.), left hand 6 inches (15.24 cm.).

Outstretched Hands: There was spasmodic pronator-supinator movement of the left hand with sometimes a few flexor movements of the left fingers.

Reflexes:	Right	Left	
Supinator	+	0	
Biceps	+	0	
Triceps	+	0	
Knee reflexes	+	+	
Ankle reflexes	+	0	
Abdominals	++	++	—one side did not tire quicker than the other
Plantars	flexor	flexor	
Clonus	0	0	

Organic reflexes normal

Gait: The patient walked as if intoxicated. The face was turned to the right and the head was inclined to the left shoulder. Irregular movements were made by the head. The patient walked on a broad base, was very ataxic, and came down on the flat of the foot. There was no difference in the attitude of the arms which hung limply by the side or were usually in readiness to help her if she fell.

On March 13, 1923, the Wassermann reaction of the blood was negative.

On March 19, an examination of the eyes was made by Dr. Williamson-Noble, who reported: Right eye: The disk outline was completely blurred; the surface was covered with numerous capillaries; the highest point was + 7 diopters, the general retina + 3 diopters. Left eye: There was a similar appearance of the left eye but with more capillaries; the highest point was + 7 diopters, the general retina was + 2.5 diopters.

On May 1, an operation was performed by Dr. Donald Armour. A large subtentorial decompression was performed, the posterior part of the foramen magnum and posterior arch of the atlas being removed. The vermis of the cerebellum was found bulging backward, but no tumor was seen on the surface of parts exposed.

The patient died the same evening.

Postmortem Examination (Dr. J. G. Greenfield).—A considerable pressure cone was present, extending as far down as the arch of the axis. The vermis was prominent and the whole of the pons, cerebellum and midbrain looked larger and was firmer than normal. When the cerebellum was lifted, a large rounded tumor mass was seen to project downward into the fourth ventricle, which it almost entirely filled. It had caused much hydrocephalus, the infundibulum being pressed down and the floor of the third ventricle being greatly distended.

On section, after hardening, the tumor was seen to have invaded the tectum opticum (region of the anterior colliculi) which it had completely destroyed. It

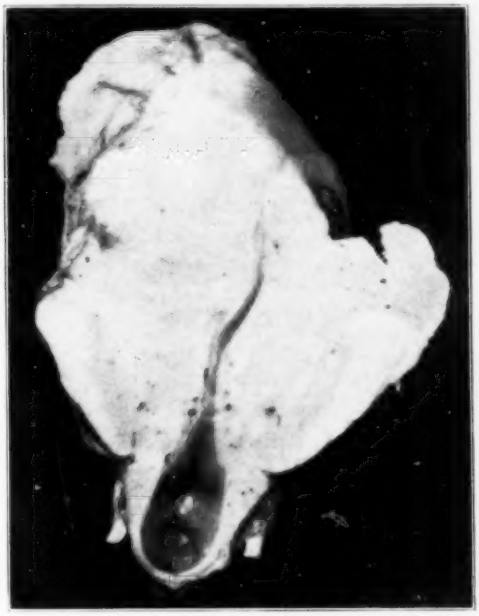


Fig. 1 (case 1).—Relationship of tumor to the oculomotor nucleus.

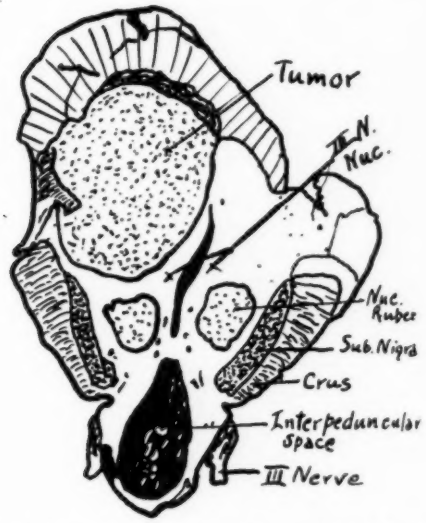


Fig. 2.—Tracing of figure 1 to clarify the anatomic relations of the tumor.

skirted the region of the third nerve nuclei, but the tegmentum, with its twin red nuclei and adjoining structures, and the crura cerebri were intact.

Histologically, the tumor was classed as a spongioblastoma multiforme.

Comment.—For our purpose in this communication, it suffices to emphasize the following clinical and pathologic features of the case:

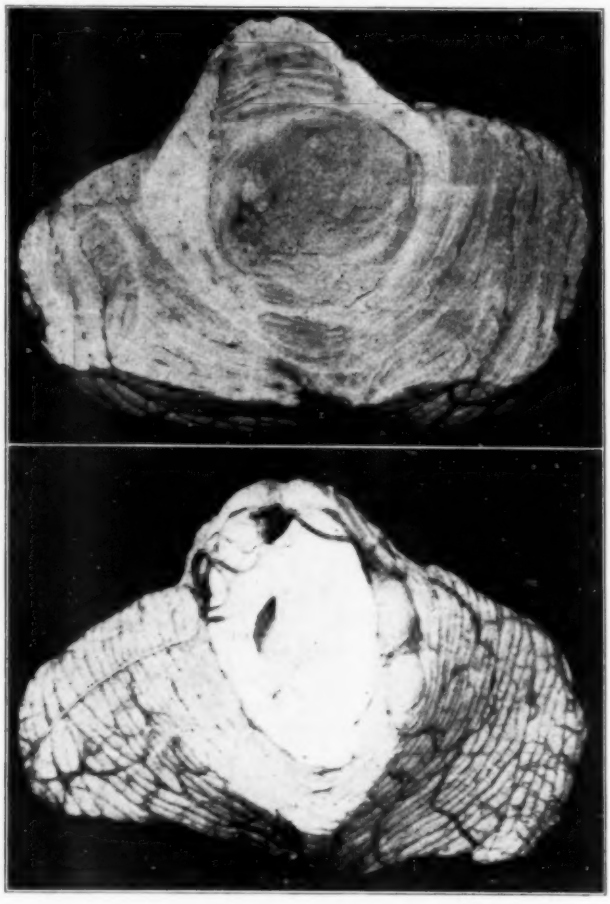


Fig. 3 (case 1).—Appearance of tumor in other sections.

A girl, aged 17, nonsyphilitic, was found to present a typical bilateral Argyll Robertson pupil sign, together with nystagmus, ophthalmoparesis, and other symptoms suggestive of a mesencephalopontocerebellar lesion. At autopsy a tumor was discovered filling the fourth ventricle, invading the upper pons and destroying the region of the tectum inclusive of both corpora quadrigemina anteriora. It was so placed that the path

for the light reflex, via these bodies, was interrupted, whereas that for accommodation, from the crura cerebri through the tegmentum to the third nerve nuclei, was intact.

CASE 2.—*History.*—E. W., a man, aged 28, was admitted to the National Hospital, Queen Square, London, on May 31, 1928, under the care of Dr. Risien Russell, and died on Aug. 7, 1928. The diagnosis was: tumor of the mesencephalon.

The symptoms began eleven months prior to admission, two weeks after an operation for varicose veins of the left leg. At that time he suffered from morning headaches and occasional attacks of vomiting. He also complained of deafness of the right ear and tinnitus. When walking he had a tendency to fall to the right, but he had never actually fallen. He was often giddy. The patient next noticed that his right thumb trembled, and gradually this shaking spread over the entire right side of the body. Following this, the right leg seemed stiff and numb, and about this time he began to have double vision and was told that he had a squint.

Fig. 4 (case 2).—Attempt of the patient to look to the right, to the left and upward.

The weakness of the right arm and leg gradually increased, and at the time he was admitted to the hospital he complained of a feeling of numbness down the whole right side of the body. He had recently had some speech difficulties, finding himself "unable to get the words out." There had been no unconsciousness or sphincter disturbance.

Examination on Admission.—Cranial Nerves: The sense of smell was normal. The visual acuity was: right 6/60, left 6/12; there was some peripheral constriction of the visual fields, more on the right than on the left. There was a well marked papilledema of both optic disks.

A pronounced limitation of conjugate upward deviation of the eyes and some defect of the outward movement of the left eye were present. When the patient attempted to look up, his eyes tended to converge. When he looked to the right a slow and coarse lateral nystagmus was obtained, which was less marked when he looked to the left. Conjugate lateral movements were good in range and downward movements were fair. The pupils were medium in size, equal, regular, and concentric. The light reflex was completely abolished in both eyes, but they reacted promptly on accommodation.

Slight hypalgesia of the right side of the face was present. There was a slight weakness of the right side of the face. The auditory acuity was approximately normal. Some hyperexcitability of the right labyrinth was present. The remainder of the cranial nerves were functioning normally.

Motor System: Hypotonia, dysmetria and dysdiadokokinesia of the right upper extremity were present with the customary tests. The presence of an intermittent tremor of the right hand and arm, both at rest and on movement, was especially noticeable. It consisted of pronation-supination alternation coupled with an abduction-adduction motion of the thumb.

Sensory System: There was relative diminution of appreciation for painful stimuli over the entire right side of the body, most marked on the face. Otherwise sensory functions were intact.

Reflexes: The right corneal reflex was sluggish. There was diminution of both abdominal reflexes, with hyperactive knee and ankle reflexes, and a doubtful extensor plantar response on the right.

Gait: The patient walked with the trunk inclined to the right, the right leg being lifted higher than the left and brought to the ground with a stamping movement.

On June 6, 1928, the Wassermann reaction of the blood was negative.

The patient left the hospital on June 12 and was readmitted on July 7.

Operation (Dr. Donald Armour, Aug. 7, 1928).—A large occipitocerebellar decompression was made. The cranial contents were obviously under much pressure, but no tumor was visible on the surface exposed.

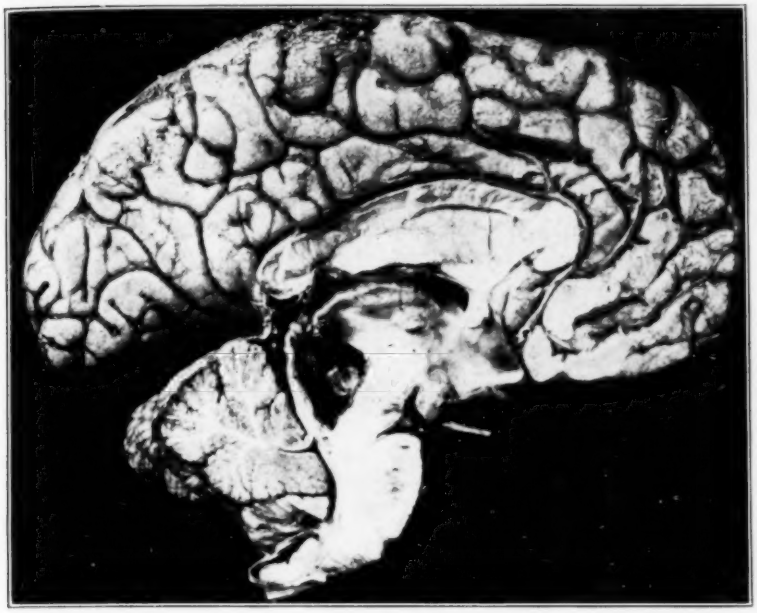
The patient died the next morning.

Postmortem Examination (Dr. J. G. Greenfield and Dr. E. O'Flynn).—When the brain was removed from the skull, a large cyst was seen occupying the interpeduncular space. Its walls were transparent, and it was filled with glairy watery fluid. The pituitary was much flattened but did not contain a tumor. When the cerebral hemispheres were separated, an apparent extension of the cyst was seen at the posterior end of the left optic thalamus. About 7 cc. of clear yellow fluid was obtained from this part of the cyst.

After the brain was hardened and suitably sectioned, the following points were determined:

There was hydrocephalus affecting both the lateral and the third ventricles. The iter of Sylvius was funnel-shaped, being dilated in its upper part but compressed from the left side at its opening into the fourth ventricle. The left midbrain contained a cyst, thin-walled in its upper posterior portion where the left anterior colliculus was flattened out. It was lined by a yellow ochreous membrane about 1 mm. thick. It contained a shriveled membranous mass attached by a thin pedicle to the posterior external wall of the cyst near the external geniculate body. As far as could be made out it had no relationship to the ventricular system. A section made through the middle of the pons and optic chiasma passed through the left red nucleus which had been pressed to the right by the cyst. When the upper end of the midbrain was sectioned a dark mass on the anterior surface was found to be a rounded tumor, with a funnel-shaped depression extending into its center from the cavity of the tumor. Around this depression the tumor was brownish, apparently as the result of hemorrhage.

The tumor extended from this level into the anterior portion of the thalamus where it ended as a rounded, fibrous-looking, hard nodule. Its end at the upper surface of the pons was similarly composed of hard tissue. In its central part it was clearly defined from the surrounding brain tissue and was fairly rounded in



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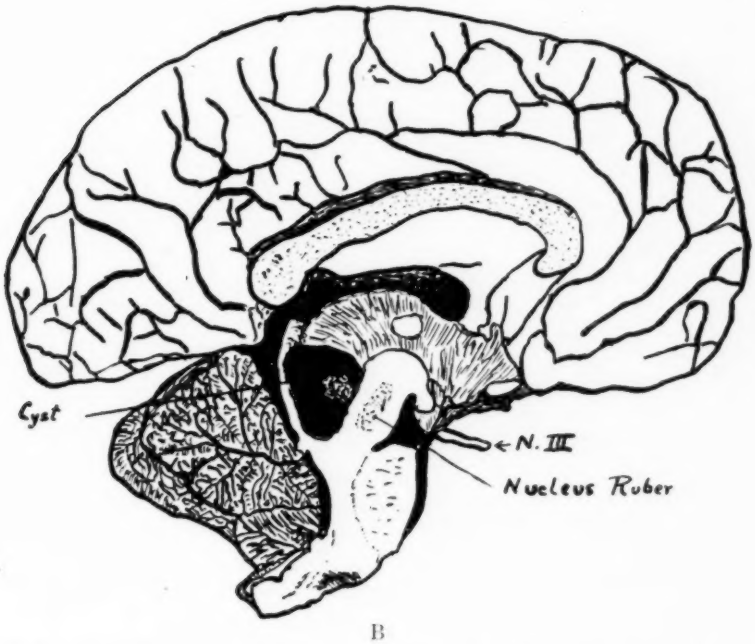

B

Fig. 5 (case 2).—*A*, median section of the brain; *B*, tracing of *A*, showing the relation of the cyst to the nucleus ruber and the third nerve.

outline. The cyst reached from the upper limit of the pons to the level of the splenium of the corpus callosum.

Histologically, the tumor was a highly vascular glioma. As can be seen from figure 5, the cystic glioma invaded and destroyed, inter alia, the whole of the region of the anterior corpora quadrigemina, whereas the crura and the tegmentum generally were intact structurally, if in part somewhat distorted.

Comment.—This case provides additional confirmation of the general truth that in nonsyphilitic conditions a tumor appropriately situated in the roof of the midbrain can produce clinically a typical Argyll Robertson pupillary sign on either side or on both sides.

EPENDYMOMAS

A CLINICAL AND PATHOLOGIC STUDY OF EIGHT CASES *

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Tumors of ependymal origin are considered rare. In an analytic study of 140 gliomas from the neurosurgical service here, eight were found to be tumors composed of cells of ependymal derivation. Five of these had their origin in the cerebrum, while three grew within the fourth ventricle. Although this is a small collection on which to base definite conclusions, certain clinical and histologic observations seem to warrant a report of these cases.

HISTOLOGIC METHODS

In the histologic study of these ependymal gliomas, the following staining methods were used: (1) hematoxylin and eosin; (2) Mallory's phosphotungstic acid-hematoxylin; (3) Bailey's neutral ethyl violet-orange G; (4) Hortega's silver carbonate for oligodendroglia; (5) Penfield's second modification of Hortega's silver carbonate for oligodendroglia; this method proved valuable in staining tissue that had been preserved in formaldehyde for a long time; (6) a modification of Hortega's silver carbonate (lithium) method for impregnating astrocytes, with sections cut from paraffin blocks (sections stained in this way made excellent preparations for microphotographs).

LITERATURE

The largest group of ependymal tumors was studied by Bailey,[1] who reported 16 of 566 gliomas analyzed in Cushing's clinic up to Jan. 1, 1927. Bailey's[2] first article on this subject appeared in 1924, but prior to that time other reports appeared in the literature. In 1902,

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* Submitted for publication, Nov. 28, 1928.

* From the neurosurgical service of Dr. Ernest Sachs at Barnes and St. Louis Childrens' Hospitals, and the neuropathologic laboratory of the Department of Surgery of Washington University School of Medicine, St. Louis.

1. Bailey, P.: Further Remarks Concerning Tumors of the Glioma Group, Bull. Johns Hopkins Hosp. **40**:354, 1927.

2. Bailey, P.: A Study of Tumors Arising from Ependymal Cells, Arch. Neurol. & Psychiat. **11**:1 (Jan.) 1924.

Saxer [3] reported five such tumors and gave excellent colored plates of the microscopic appearances. In the same year, Mallory [4] reported three gliomas of ependymal origin. In 1907, Spiller [5] described an ependymal tumor that arose from the lining of the fourth ventricle, this case being of particular interest in that a neoplasm of identical structure was found in the lower portion of the thoracic cord; it was believed by him to be, in all probability, a metastatic growth. Spiller also referred to another ependymoma reported by himself in 1903.[6] Roussy, Lhermitte and Cornil,[7] in 1924, described a group of gliomas under the term ependymogliomas which in the opinion of Penfield [8] belong in this category. Hirsch and Elliott [9] reported two ependymomas in 1925, and in the same year Orlandi [10] described one. Silverberg [11] reported a similar tumor in 1926.

In order to get a clear picture of present views of ependymal gliomas, a brief conception based on recent literature is desirable. Grossly, these tumors are usually described as pale, encapsulated, nodular and firm, with a tendency to calcium deposition and cyst formation. By far the greatest number of these have been located in the posterior fossa in close proximity to the fourth ventricle. Of the twelve tumors reported by Bailey and Cushing [12] up to 1926, nine grew in this region, and the great majority of ependymal growths reported by other writers have likewise arisen in this general locality. Bailey and Cushing have drawn attention to a valuable differential diagnostic point between these tumors and another type which is encountered more frequently in the midcerebellar region—the medulloblastomas. The

3. Saxer, F.: Ependymepithel, Glioma und epithiale Geschwülste des Centralnervensystems, Beitr. z. path. Anat. u. z. allg. Pathol. **32**:276, 1902.

4. Mallory, F. B.: Three Gliomata of Ependymal Origin: Two in the Fourth Ventricle, One Subcutaneous Over the Coccyx, J. M. Research **8**:1 (June) 1902.

5. Spiller, W. G.: Gliomatosis of the Pia and Metastasis of Glioma, J. Nerv. & Ment. Dis. **34**:297, 1907.

6. Spiller, W. G., and Hendrickson: A Report of Two Cases of Multiple Sarcomatosis of the Central Nervous System and One Case of Intramedullary Primary Sarcoma of the Spinal Cord, Am. J. M. Sc., July, 1903, p. 10.

7. Roussy, G.; Lhermitte, J., and Cornil, L.: Essai de classification des tumeurs cérébrales, Ann. d'anat. path. med.-chir. **1**:333, 1924.

8. Penfield, W.: Principals of Pathology of Neurosurgery, Nelson Loose Leaf Living Surgery, vol. 2.

9. Hirsch, E. F., and Elliott, A. R.: Ependymomas of the Lateral and Fourth Ventricles of the Brain, Am. J. Path. **1**:627, 1925.

10. Orlandi, H.: Papiloma ependimaria del IV ventriculo, Clin. y. lab. rev. quincen. de espec. méd., Zaragoza, vol. 4, no. 27.

11. Silverberg, E.: Neuroblastoma and Neuro-Epithelioma, Virchows Arch. f. path. Anat. **260**:251, 1926.

12. Bailey, P., and Cushing, H.: Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.

ependymomas tend to grow down into the spinal cord while, in their experience, the medulloblastomas rarely do. This was a characteristic of the case Spiller[6] reported in 1903, in which the tumor grew down along the cord from the fourth ventricle to the level of the sixth cervical vertebra.

Ependymal tumors are said to be of slow growth. Bailey and Cushing [12] noted that they grow much more slowly than might be expected from the unusually short duration of symptoms prior to hospitalization. Bailey [2] mentioned the case of a woman in excellent health eleven years after a cerebellar tumor had been exposed but not disturbed. This, in his opinion, was undoubtedly an ependymoma. One of the tumors reported by Mallory,[4] which arose in the midline of the back in the coccygeal region, lay dormant for twenty-five years and then suddenly started to grow rapidly. The firmness, definite encapsulation, tendency to calcium deposition and cyst formation have been mentioned as evidence that they grow slowly.

Microscopically, ependymal neoplasms were at one time divided by Bailey and Cushing [12] into two groups: (1) those composed chiefly of the less differentiated spongioblasts, which they called ependymoblastomas; (2) those in which the tumor cell type was predominantly a more adult polygonal cell, devoid of processes, which they designated ependymomas. In a later communication,[1] Bailey classified all ependymal tumors as ependymomas, as the clinical behavior of the two types seemed too similar to justify grouping them separately. Histologically, little has been added to the excellent description given by Mallory in 1902.[4] Sections are usually described as very cellular, with many thin-walled vascular channels. The cells are closely packed, containing single large oval or spherical vesicular nuclei with a few chromatin particles, and not infrequently with single distinct nucleoli. Mitotic figures are extremely rare. The cytoplasm of the adult cells is abundant and coarsely granular, and the cell itself is polygonal. The cytoplasm stains poorly with eosin but is well demonstrated with Bailey's ethyl violet-orange G or with Mallory's phosphotungstic acid-hematoxylin. The primitive ependymal spongioblasts occur in variable numbers. They are described as tadpole-shaped cells with single heavy tails, the ends of which terminate in the vicinity of the walls of the blood vessels. There may be no definite arrangement of the cells in these tumors, but if the less differentiated ependymal spongioblasts predominate a definite architecture is noted, which even under low magnification is characteristic. The thin-walled vascular channels stand out sharply, owing to a clear zone which encircles them. The clear collars about the vessels are in turn surrounded by closely packed tumor cells. When such sections are stained with appropriate methods, these "clear zones" are seen to be filled with the processes of the embryonic cells.

Mallory [4] was the first to suggest that the small clusters of deeply staining chromatin granules found in the cytoplasm of ependymal cells differentiated these gliomas from others, and he expressed the opinion that perhaps all ependymal gliomas "were definitely characterized in this manner." These minute granules surrounded by clear halos are known as blepharoplasts and are said to be the remnants of small chromatin particles at the base of the cilia which are present in normal ependymal cells. They are well demonstrated with neurofibrillar stains. Bailey pointed out that the presence of these granules in the cells of the brain tumor is not sufficient in itself to warrant a diagnosis of ependymoma, for, as he has shown, certain pineal and subependymal cells have similar granules in their cytoplasm.

In a consideration of the clinical aspects of these growths, Bailey's and Cushing's [12] experience is of particular importance. Ependymomas are most commonly tumors of childhood. Since their favorite site of origin is in close proximity with the fourth ventricle, attempts at removal are precarious, for manipulation of these tumors invariably causes respiratory embarrassment. The symptoms presented by these growths in the posterior fossa are both general and focal. The localizing features are a mixture of cerebellar and fourth ventricle symptoms. Bailey stresses the early vomiting that occurs in these cases and explains it on the basis of pressure on the vagus center. He believes that suboccipital pain of which these patients frequently complain is due to the downward extension of the tumor into the spinal canal.

REPORT OF CASES

CASE 1.—*Ependymoma within the Lateral Ventricle.*

R. A., a man, aged 17, was admitted to Barnes Hospital on Dec. 27, 1916, with a history of headache, failing memory and generalized convulsions for four months. There were: a choking of both disks, hypesthesia of the right cornea, right facial weakness, ataxia and adiadokokinesis of the right hand and arm. Roentgenograms of the skull showed marked convolutional atrophy of the inner table.

Operation.—On Jan. 4, 1917, Dr. Sachs performed a right subtemporal decompression. There was no relief of symptoms, and, owing to the continued increasing pressure, a second decompression was done on the opposite side. There was no improvement, and the patient died shortly thereafter.

Postmortem Observations.—Autopsy revealed a nodular, pale gray, firm tumor attached in the right anterior horn; the growth filled both ventricles of the cerebrum. It measured 8.6 by 5 by 6.5 cm. Two small cysts presented on the surface.

Microscopic sections showed a cellular and moderately vascular lesion. The cells were crowded together in no particular arrangement. They possessed large, spherical, vesicular nuclei. No mitotic figures were seen. The vessels were thin-walled, but plainly outlined by clear spaces about each one. Hortega's silver carbonate (lithium) stain for demonstrating astrocytes used with paraffin-embedded sections showed that a great number of the cells were typical ependymal spongio-

blasts, the long processes of which formed a thick tangled network of fibers. Blepharoplasts were demonstrated best in the sections stained with neutral ethyl violet-orange G. In the same sections the polygonal outline of the cells was best demonstrated.

CASE 2.—*Ependymoma of the Fourth Ventricle.*

H. K., a male child, aged 16 months, entered the Children's Hospital with a history of marked irritability, gradual loss of the use of the extremities and inability to raise the head. The mother had noted a rapid increase in the size of the child's head for three months before admission. Besides a choking of both disks, examination revealed spasticity of both lower extremities with bilateral ankle clonus. Phenolphthalein injected into the lateral ventricle showed an obstruction of the flow of the cerebrospinal fluid.

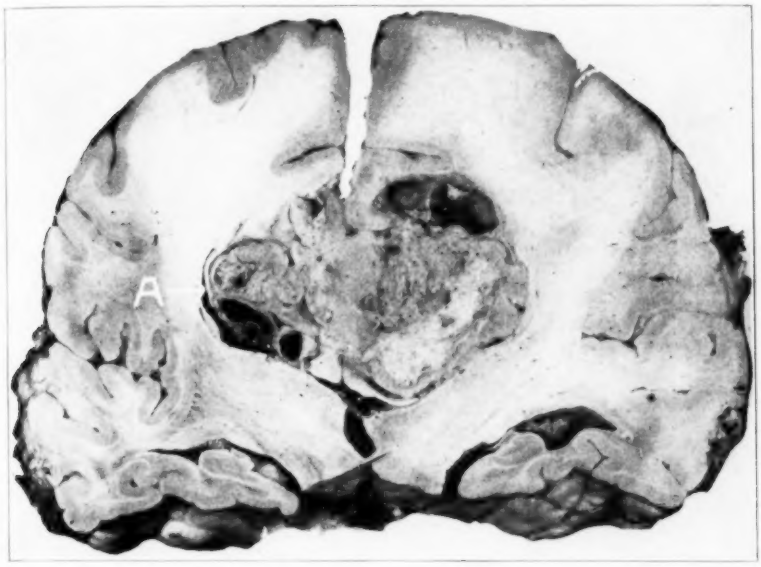

Fig. 1 (case 1).—*A*, septum pushed way over to the left by the tumor which has its attachment in the roof of the right ventricle. It should be noted that the cysts are all on the surface of the tumor.

Operation.—On Nov. 3, 1917, Dr. Sachs did a cerebellar exploration and a large nodular tumor was exposed; it filled the basal cistern and extended downward over the cervical cord. No attempt was made to remove the tumor. Immediately after the cerebellar decompression the symptoms subsided, but by March, 1918, the patient had ceased talking and the movements of the extremities had again become awkward. On May 16, 1918, Dr. Sachs performed a second cerebellar operation and part of the tumor was removed piece-meal. The patient died on the second day after the operation.

Autopsy.—Grossly, the lesion was composed of various sized, pale gray, fairly soft nodules which completely filled the basal cistern, almost enveloped the brain stem and extended down over the cervical cord. The tumor apparently arose from the posterior medullary velum. On section the surfaces of the nodules had a mottled gray and pink appearance.

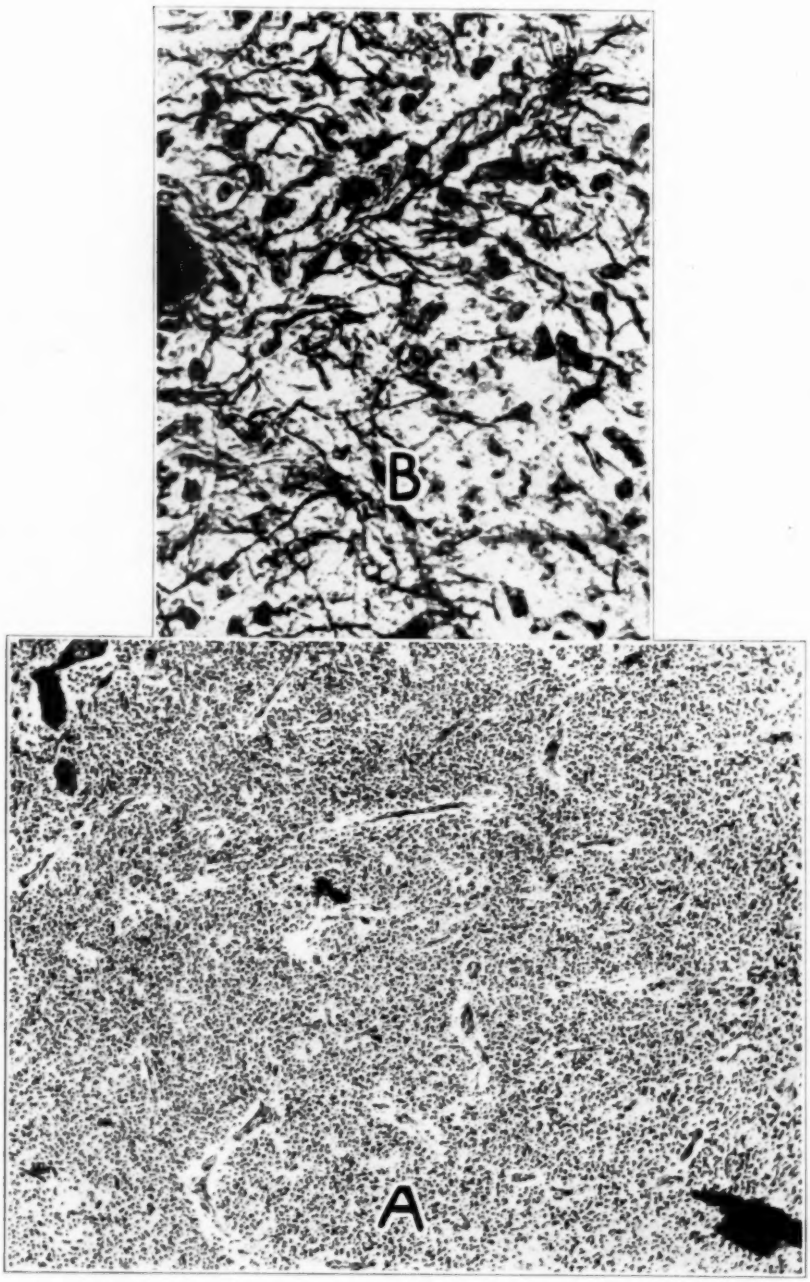

Fig. 2 (case 1).—*A*, the "clear spaces" about the vascular channels. Ethyl violet-orange G stain; × 88. *B*, scattered throughout the sections are found numerous young ependymal cells. Hortega's silver carbonate (lithium) stain.

Histologically, the tumor was moderately cellular with considerable intracellular stroma. The vascularity was variable in different sections. Immediately around the vessels were clear zones. These cells were more or less closely packed with a tendency toward pseudoroset formation. Little could be ascertained concerning the nature of the cells with eosin and hematoxylin staining other than that they contained large oval vesicular nuclei with no mitotic figures. Silver carbonate stains showed that a greater number of the cells were typical ependymal spongioblasts the long wirelike tails of which streamed for considerable distances. In the ethyl violet-orange G and phosphotungstic acid preparations, clusters of tiny granules were seen in the cytoplasm of the polygonal cells.

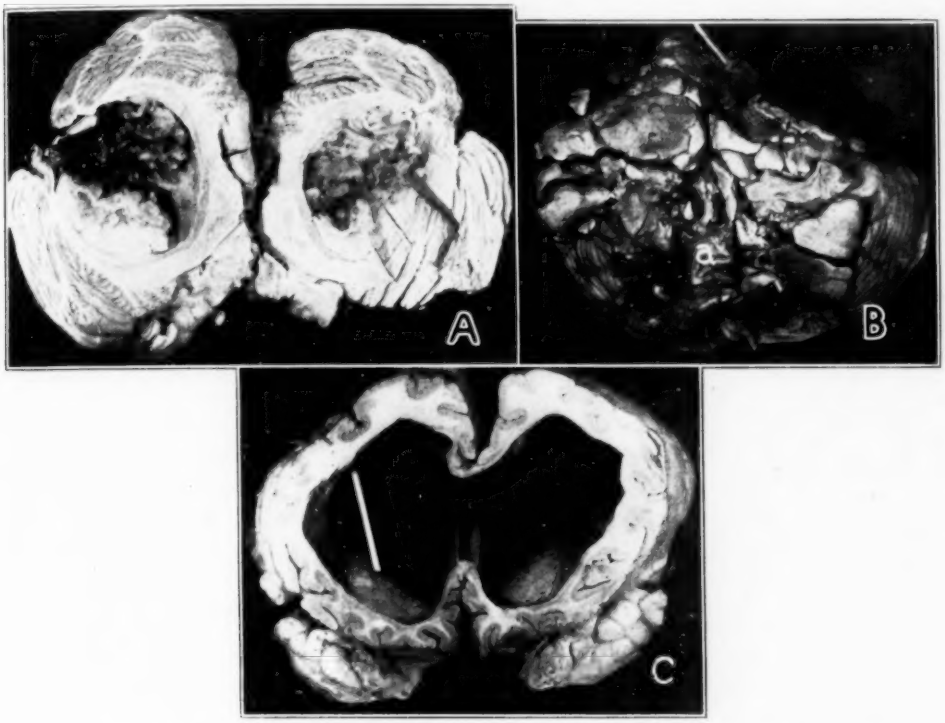

Fig. 3 (case 2).—*A*, the tumor has compressed enormously both the cerebellar nuclei and the pons. *B*, anterior surface of the brain stem and cerebellum showing how extensively the tumor has enveloped the brain stem and upper cervical cord. *C*, note the extensive dilatation of the cerebral ventricles. *a* is the brain stem.

CASE 3.—*Ependymoma of the Fourth Ventricle.*

J. J., a boy, aged 7, was admitted to the Children's Hospital on April 5, 1921, complaining of headaches, frequent attacks of vomiting, jerky respirations and a constant nodding of the head. These symptoms began one month before entry following the cessation of a chronic discharge from the left ear which had continued for three years. Positive neurologic observations were: bilateral choked disk, slight facial paresis on the right side, a tendency to hold the head tilted

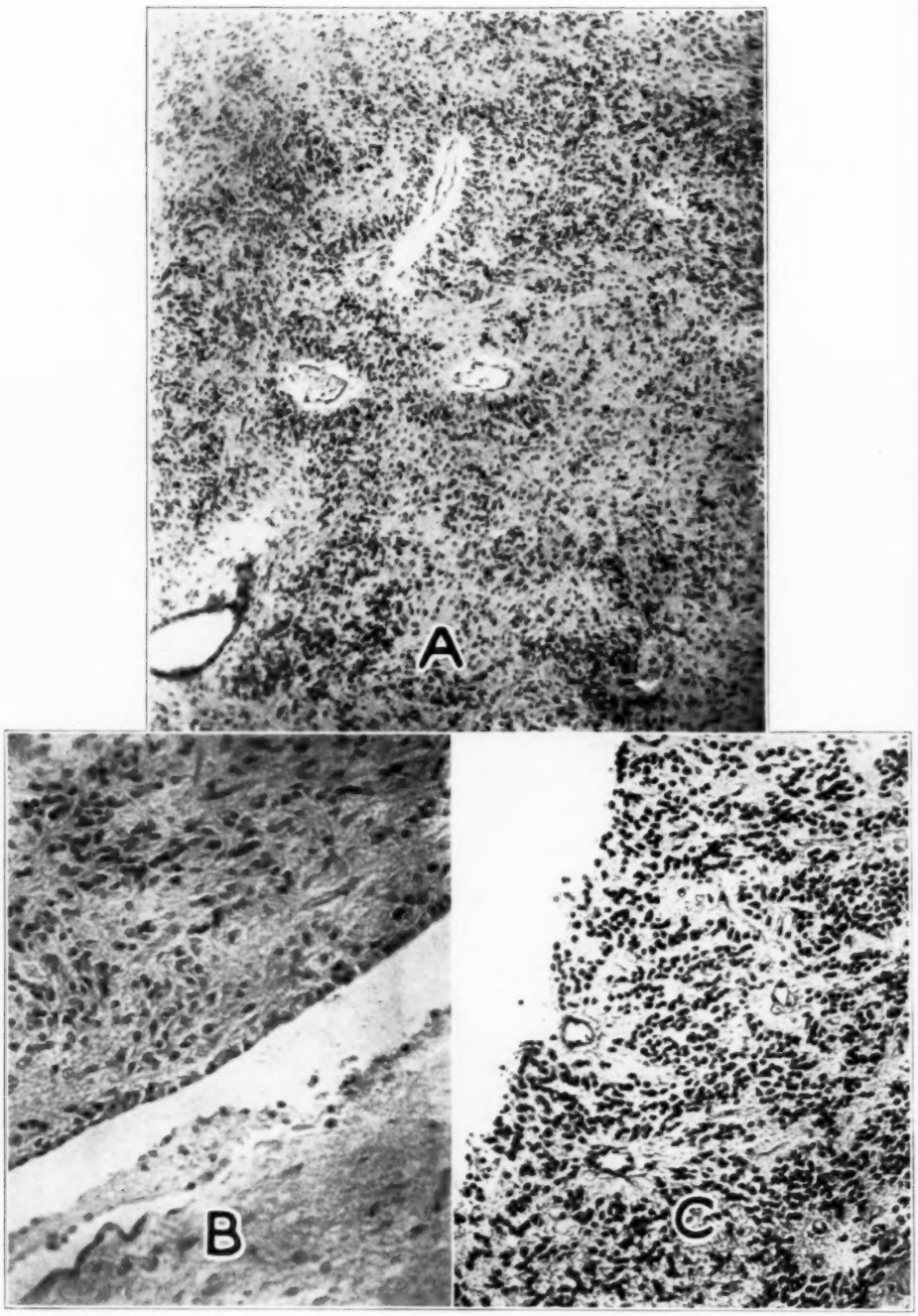

Fig. 4 (case 2).—*A*, the palisading of the cells about the blood vessels is strikingly shown (section from portion of tumor shown in figure 3 *A*). Hematoxylin and eosin stain; × 88. *B*, another area of figure 3 *A*, showing a single layer of ependymal cells demarcating the tumor from cerebellar tissue. Hematoxylin and eosin stain; × 170. *C* (section from one of the nodules illustrated in figure 3 *B*); this part of the tumor was much more cellular, showing pseudoroset formation.

toward the left shoulder, slight ataxia of the left arm, absent patellar and ankle reflexes on the right and a cerebellar gait. X-ray pictures of the skull showed marked separation of the sutures and convolutional markings.

Operation.—On April 8, 1921, Dr. Sachs trephined the skull and explored the cerebellum with a ventricle needle for a possible abscess of the brain. None was encountered and a left decompression was done. On April 15, 1921, there having been no relief of the intracranial symptoms, a right-sided decompression was performed. There was still no relief, and on May 2, 1921, Dr. Sachs did a cerebellar exploration which uncovered a glioma involving the inner third of both cerebellar

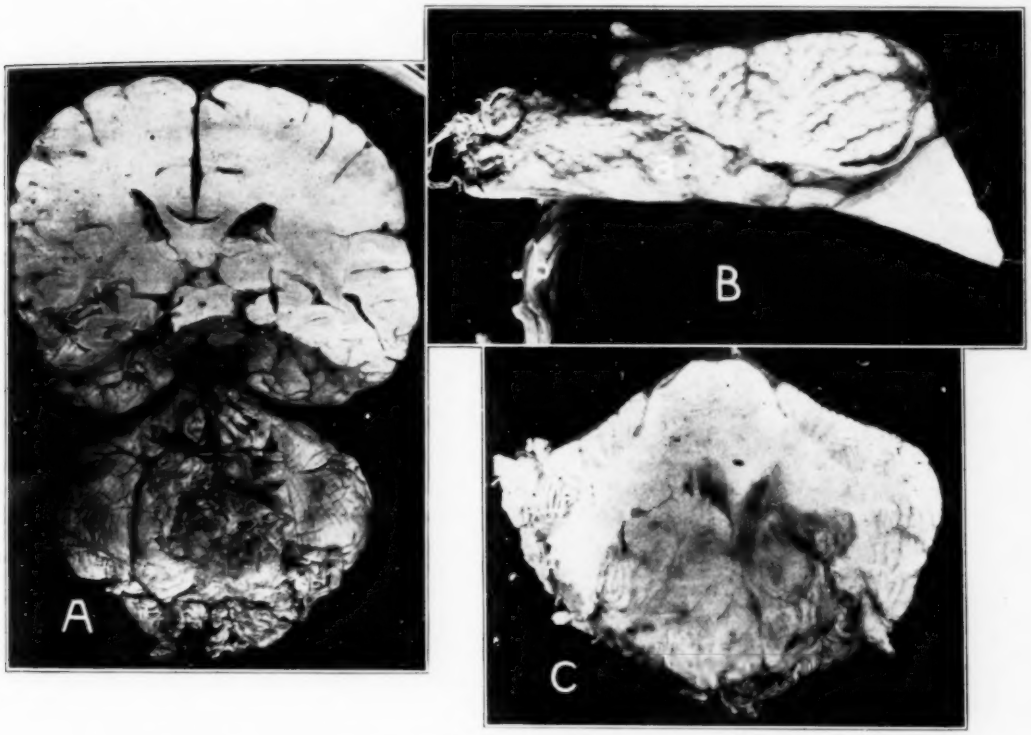

Fig. 5 (case 3).—The ventricles are not dilated. The inferior surface of the cerebellum shows the tumor sharply demarcated. The cerebellar hemispheres are spread wide apart. *B*, downward extension of tumor; (*a*) tumor; (*b*) spinal cord. *C*, obstruction of fourth ventricle not complete. Tumor has grown downward and backward. Abundance of fibrous tissue well seen in the tumor.

hemispheres, and extending down into the fourth ventricle to the foramen of Magendie with obliteration of the basal cistern. No attempt was made to remove the tumor. Manipulation caused symptoms of medullary embarrassment. Following the release of the pressure, the patient improved and was discharged on May 20, 1921. Within three months, his condition became desperate, and on Sept. 9, 1921, he was readmitted. An immediate operation was performed by Dr. Sachs, and partial removal of the tumor was accomplished. Radium

emanation needles were inserted into the remaining tumor. Soon after the sutures were removed from the operative wound, a cerebrospinal fistula developed. The postoperative course was a rapid, down-hill one, and death occurred on Oct. 6, 1921.

Autopsy.—The cerebrum showed little alteration; the lateral and third ventricles were not dilated. A firm nodular, brown tumor obliterated and replaced the tonsils and the inferior vermis, spreading apart the cerebellar hemispheres. The cut surface of the tumor was mottled brown, pink and gray. The tumor originated from the posterior medullary velum and filled the fourth ventricle. It measured 6 by 5 by 2.5 cm.

Microscopic sections showed a cellular tumor, the cells being scattered about with no definite pattern. Little could be ascertained as to the nature of the cells from the eosin and hematoxylin stains other than that the cells contained large oval or spherical vesicular nuclei. There were many thin-walled blood channels which stood out plainly because they were surrounded by clear zones. There were no mitotic figures in the sections studied. Small calcium plaques were seen scattered among the cells. In sections stained with Bailey's ethyl violet-orange G, many polygonal cells were seen in the cytoplasm, some of which were blepharoplasts. The silver carbonate preparations showed a predominance of young ependymal cells the long wavy processes of which interlaced or ran in parallel lines, giving an appearance somewhat similar to that of the bands of collagen seen in fibroblastic tumors of the cranial nerves.

CASE 4.—*Ependymoma of the Right Parietofrontal Region.*

K. S., a girl, aged 9, entered the Children's Hospital on Feb. 13, 1922, complaining chiefly of double vision, which had been present since July, 1921. In August, headaches and vomiting began. In October, vision began to fail, and by December she was practically blind. About the time when vision began to fail, a motor weakness of the entire left side manifested itself. In January, failing memory was first noted.

Examination.—There were: secondary optic atrophy of both nerve heads, left sixth nerve weakness, left facial weakness, and complete left hemiplegia. X-ray pictures of the skull showed marked convolutional atrophy. Just to the right of the midline, about 3 cm. below the vertex, were three circular calcified nodules in the brain substance.

Operation.—On Feb. 15, 1922, a right-sided craniotomy was done by Dr. Sachs and a large gray tumor was seen extending from the motor area far forward into the frontal region. When an attempt was made to puncture the posterior horn of the ventricle a cyst was encountered from which 90 cc. of yellow fluid was removed. The patient's condition was such that the operation was stopped. Five days later, the wound was reopened. Again the large cyst encountered at the first operation was evacuated. A large tumor was removed, after which a cyst in the region of the corona radiata, approximately 4 cm. in diameter, was seen, and its lining was treated with Zenker's fluid. The tumor was lobulated, white and firm. It measured 7.5 by 5.5 by 3.5 cm. At one end were four small, thin-walled cysts, all of which contained yellow fluid. Roentgen treatments were given. On March 10, 1922, the patient was discharged in an improved condition, but she was still ataxic. Observation in October, 1922, showed marked improvement in the child's mental condition. Vision was practically gone, and there was still evidence of involvement of the pyramidal tract. On Jan. 1, 1923, the child was readmitted to the hospital after a rather sudden development of dizziness, aphasia and bilateral involvement of the sixth nerve. On Jan. 5, 1923, Dr. Sachs reexplored the tumor

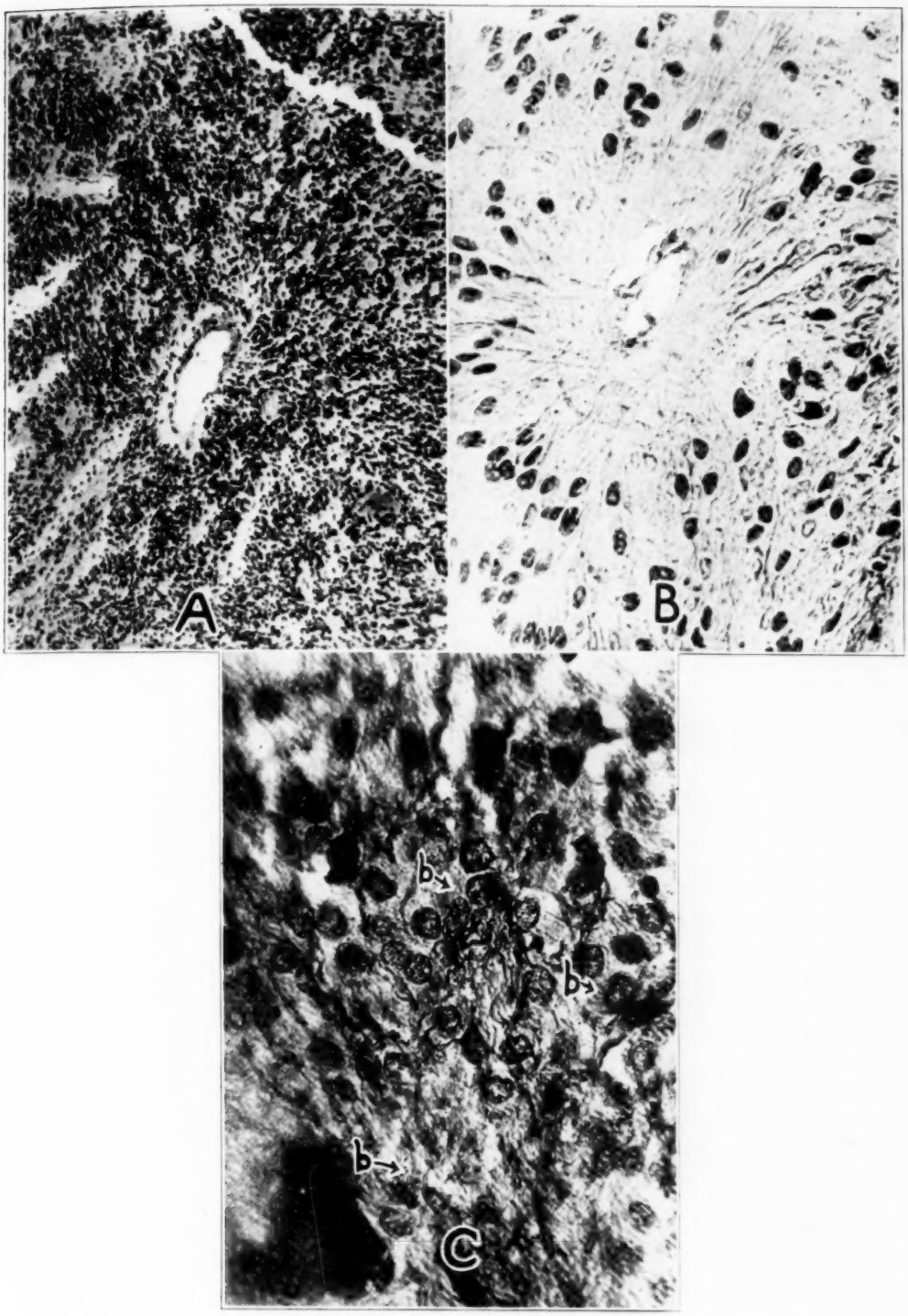

Fig. 6 (case 3).—*A*, fixation of the tissue in Zenker's solution may account for the depth of staining of the cells. Hematoxylin and eosin stain; × 88. *B*, cell processes which fill the "clear spaces" about the blood vessels. Ethyl violet-orange G stain; × 580. *C*, the large polygonal cells which characterize these tumors show blepharoplasts (*b*). Ethyl violet-orange G stain; × 700.

area, and an encapsulated tumor with the same gross attributes as those noted at the first operation was removed. This growth weighed 270 Gm. The patient became pulseless and died on the operating table.

Postmortem Observations.—Autopsy showed that all of the tumor had been removed, the adjacent brain showing some gelatinous degeneration.

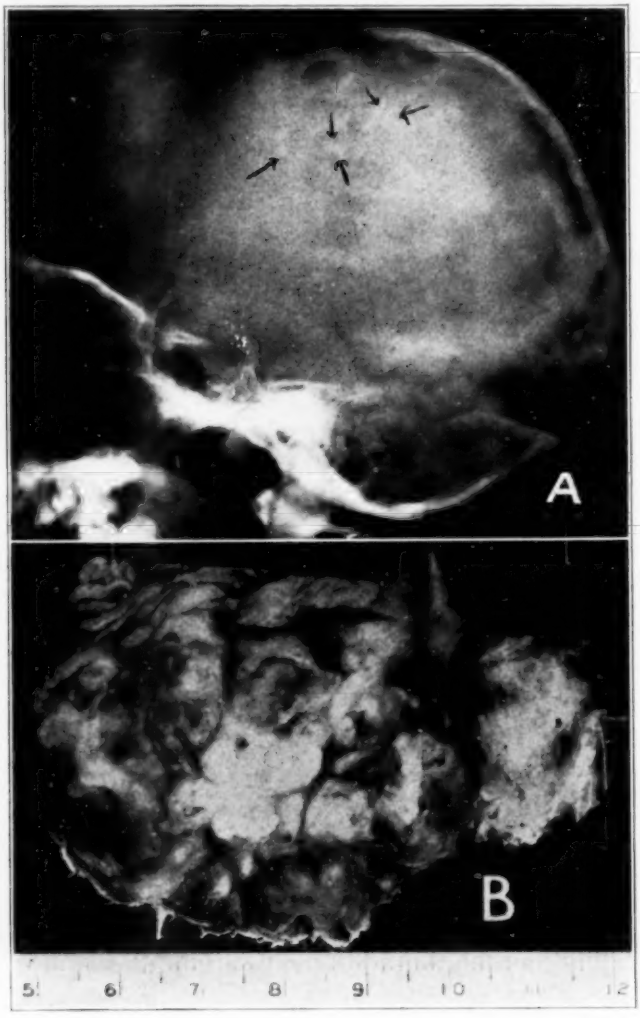

Fig. 7 (case 4).—*A*, the arrows indicate the region of calcified areas. *B*, encapsulated tumor removed at first operation.

Histologically, the tumor was cellular, the cells having fairly typical chromatic, oval or spherical nuclei which in many places were disposed in radiating rows around central vascular channels; the latter were surrounded by clear collars. No mitotic figures were seen. The majority of the cells were typical ependymal spongioblasts and were demonstrated best with silver carbonate stains. Some of

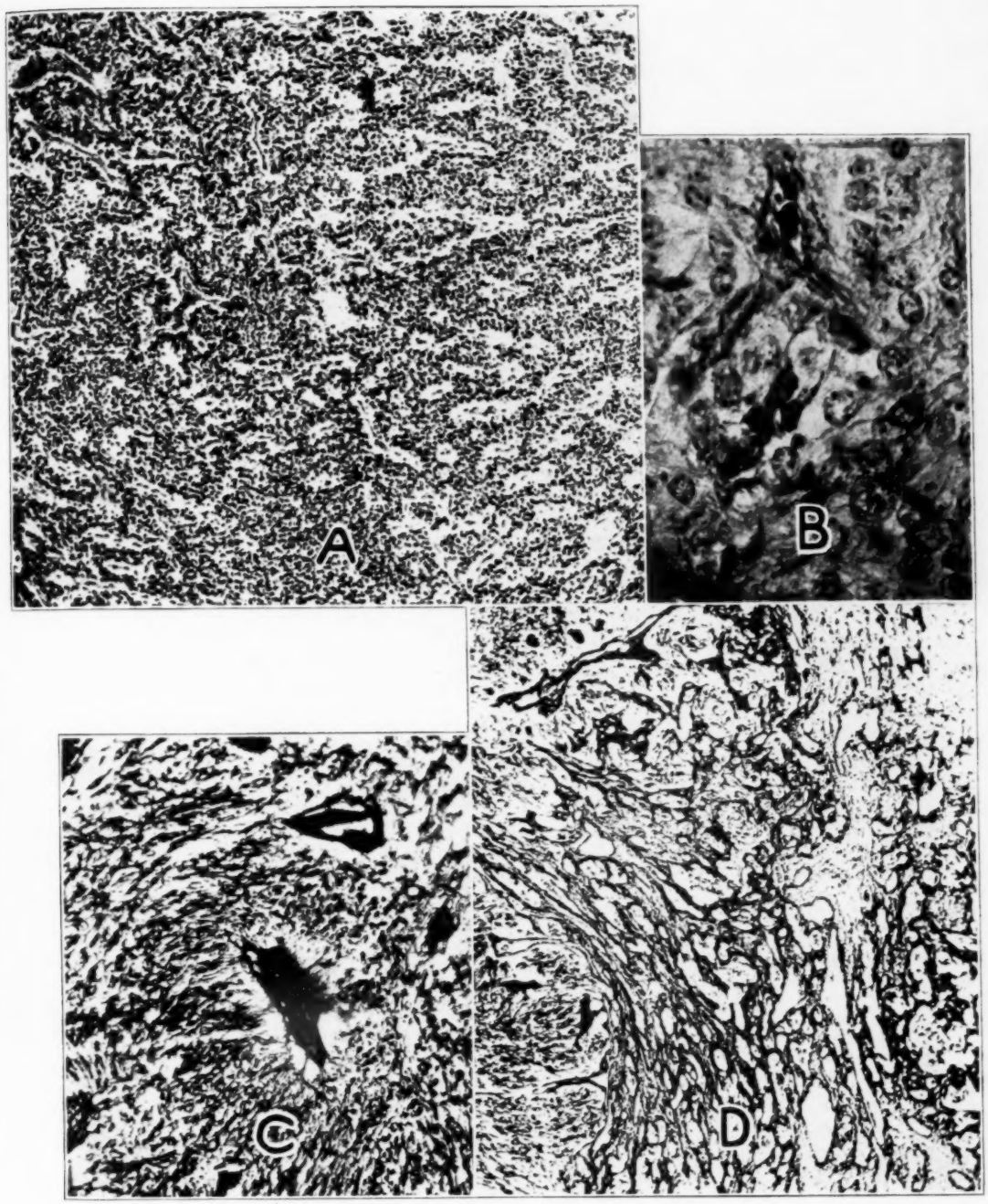

Fig. 8 (case 4).—*A*, the clear perivascular spaces make the blood vessels stand out clearly. Hematoxylin and eosin stain; × 88. *B*, showing blepharoplasts in the cytoplasm of the polygonal tumor cells. Ethyl violet-orange G stain; × 600. *C*, the processes of the ependymal spongioblasts radiating to the vessel wall are well shown here. Silver carbonate (lithium) stain; × 170. *D*, sections made from the tumor removed at the second operation. The strikingly different picture is due to the abundance of fibrous tissue which was the result of roentgen therapy. Silver carbonate (lithium) stain; × 88.

the tumor cells were polygonal and had abundant cytoplasm. Blepharoplasts were seen in this cytoplasm. Heavy strands of connective tissue were seen in many sections of the tumor removed at the second operation when stained with routine methods. This defense fibrous tisue might be the result of irradiation.

CASE 5.—*Ependymoma of the Fourth Ventricle.*

B. H., a woman, aged 34, came to the hospital on Oct. 31, 1927, complaining of dizziness and staggering with a tendency to pitch forward and to the right. The onset of the symptoms had occurred eighteen months prior to admission. Projectile vomiting was of fourteen months' duration. For six months she had had occipital and frontal headaches accompanied by a roaring in the occipital region. Vision in the right eye had been failing for five months. The left eye had been enucleated in September, 1927, following an injury.

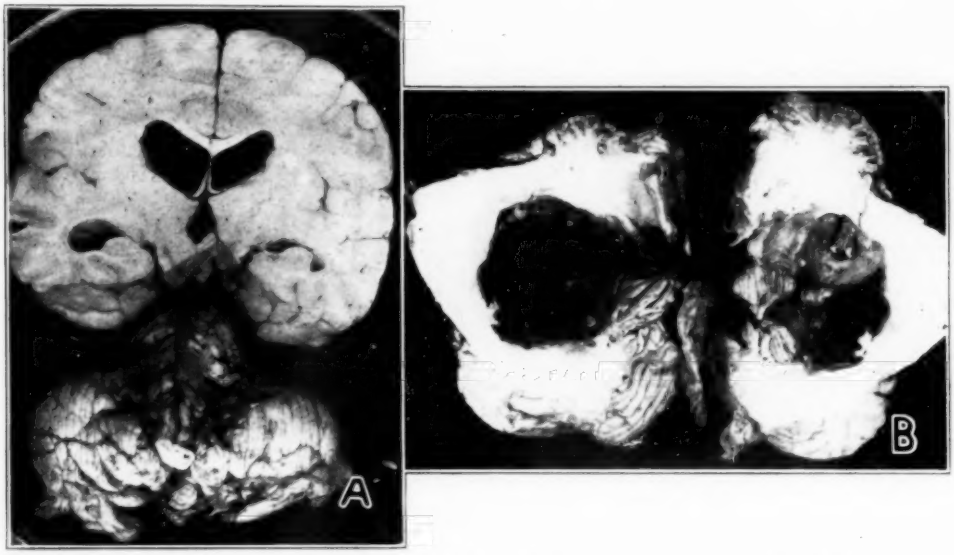

Fig. 9 (case 5).—*A*, dilated ventricles and the pressure cone on the inferior surface of the cerebellum. *B*, tumor nearer the surface at autopsy than at operation as a result of decompression.

Neurologic Examination.—There were: a high grade of choked disk; a distinct staggering gait with a tendency to fall to the right, ataxia of the extremities and tremor of the tongue, increased reflexes and bilateral ankle clonus. X-ray pictures of the skull showed a destruction of the dorsum sellae and a convolutional atrophy of the inner table of the bone. Pneumoventriculograms showed a dilatation of both the lateral and the third ventricles.

Operation.—On Nov. 4, 1927, Dr. Sachs explored the cerebellum, but no lesion was seen. A needle inserted into the left cerebellar hemisphere at a depth of 5 cm. encountered resistance. The postoperative course was stormy; a local abscess developed at the lower end of the operative wound in the fourth week; symptoms of meningitis became manifest on December 17, and on the next day drainage of the cistern was instituted. On January 1, the patient died.

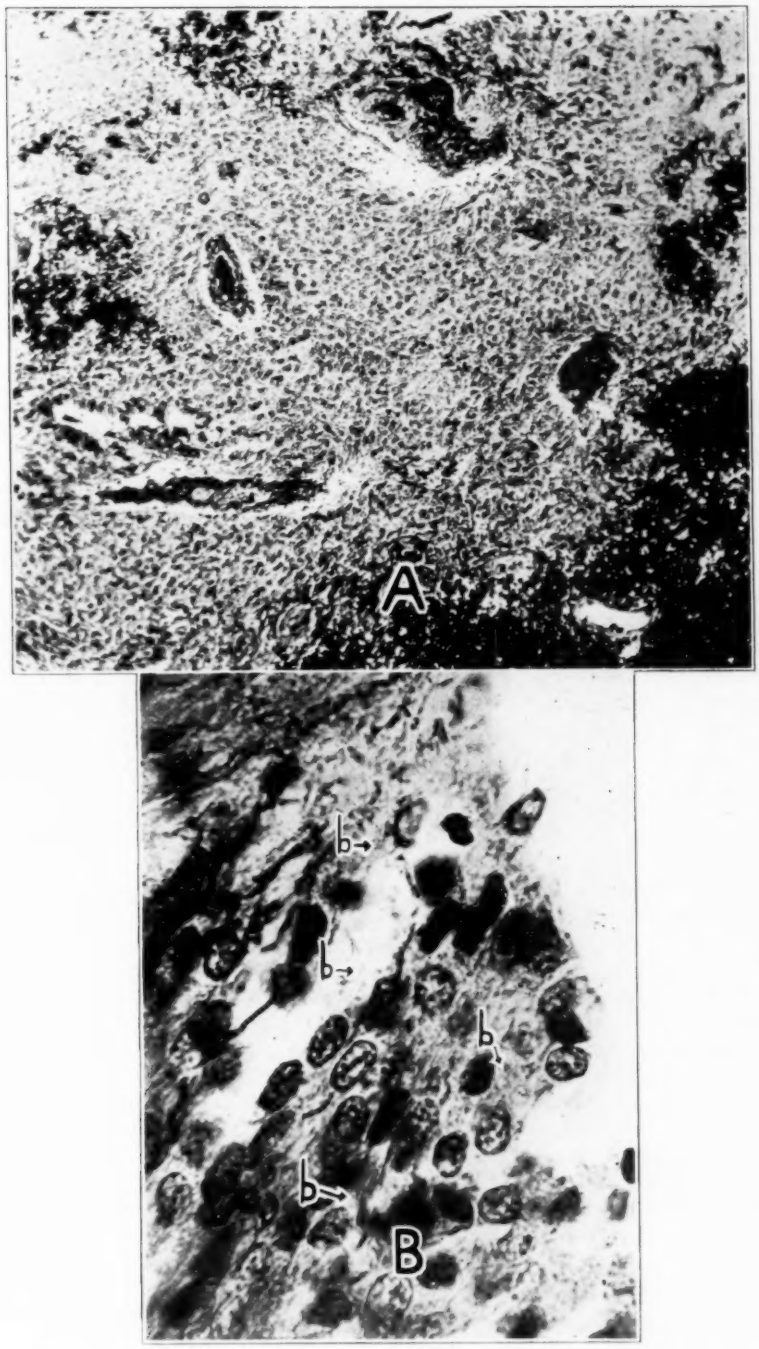

Fig. 10 (case 5).—*A*, the vesicular nature of these cells is very clear. Ethyl violet-orange G stain; × 88. *B*, blepharoplasts (*b*).

Autopsy.—The convolutions of the brain were markedly flattened. Part of the cerebellum was herniated into the foramen magnum. The third and lateral ventricles were moderately dilated. A hemorrhagic tumor about the size of a hen's egg almost replaced the median lobe of the cerebellum. It was attached to the floor of the fourth ventricle. The cut surface was mottled dark red and gray.

Histologically, in sections stained with hematoxylin and eosin, the tumor was seen to be very cellular with many blood vessels. The cells had single large oval vesicular nuclei, were not arranged in any specific pattern and were closely packed. In the cytoplasm of these cells, blepharoplasts were seen. There was considerable hemorrhage throughout each section studied.

CASE 6.—*Ependymoma of the Frontoparietal Region.*

E. H., a girl, aged 11, was admitted to the Children's Hospital on Jan. 23, 1928, complaining of frontal headaches and vomiting of two months' duration. She had had an internal strabismus of the left eye three weeks before. At the same time there was noted a tendency to staggering. There was some visual disturbance of one month's duration.

Examination.—There were: bilateral choked disk, lateral nystagmus, paresis of the left sixth nerve, ataxia of both arms and legs, adiadokokinesis of both hands, absent knee and ankle reflexes and staggering gait with a tendency to walk with a broad base. Roentgen examination of the skull showed, besides convolutional markings, circumscribed calcifications in the frontoparietal region of the brain. Because the roentgen observations threw doubt on the clinical symptoms, injection of air was attempted, but only a few drops of cerebrospinal fluid were obtained, and no air was put in.

Operation.—On Jan. 30, 1928, a left frontal bone flap was turned down by Dr. Sachs. On opening the dura, a grayish-red circumscribed lesion was seen on the surface in the facial center. The pia was split, and the tumor was removed. Examination showed another mass as large extending down under the motor cortex. This circumscribed mass was also removed. The patient made a rapid postoperative recovery and was discharged on Feb. 14, 1928. In October, the patient was reported as perfectly well.

Histologically, the tumor removed at the operation was composed of closely packed cells with large oval and spherical vesicular nuclei. Every area examined under low magnification in hematoxylin and eosin sections had a striking appearance. The manner in which the closely packed cells lay at some distance from the vascular channels was distinctive. The chromatin of the nuclei was condensed in places, giving the cells the appearance of having a distinct single nucleolus. An abundance of ependymal spongioblasts was seen in the silver carbonate sections, their wavy wirelike processes trailing for long distances and forming intricate meshes, while in other places they were disposed in parallel columns. Small granules, varying in number from two to five in a cluster, were also seen in these silver preparations. Since each group was surrounded by a clear halo and the arrangement of these granules was so like that seen with other stains in ependymal cells, they were doubtless blepharoplasts. In the ethyl violet-orange G preparations these granules were easily identified. A few adult polygonal cells were seen in these sections.

CASE 7.—*Ependymoma Located in the Left Occipital Lobe.*

P. S. J., a boy, aged 16, was admitted to Barnes Hospital on March 19, 1928, complaining of left occipital headache accompanied by occasional vomiting attacks and an inability to see objects on the right. The onset of the illness took place in the fall of 1927.

Neurologic Examination.—There were: double choked disks with retinal hemorrhages, right homonymous hemianopic field defect, and vision in the right eye so far gone that only a small sector was outlined. Roentgenograms of the skull showed marked intracranial pressure changes and an area of calcification in the occipital region.

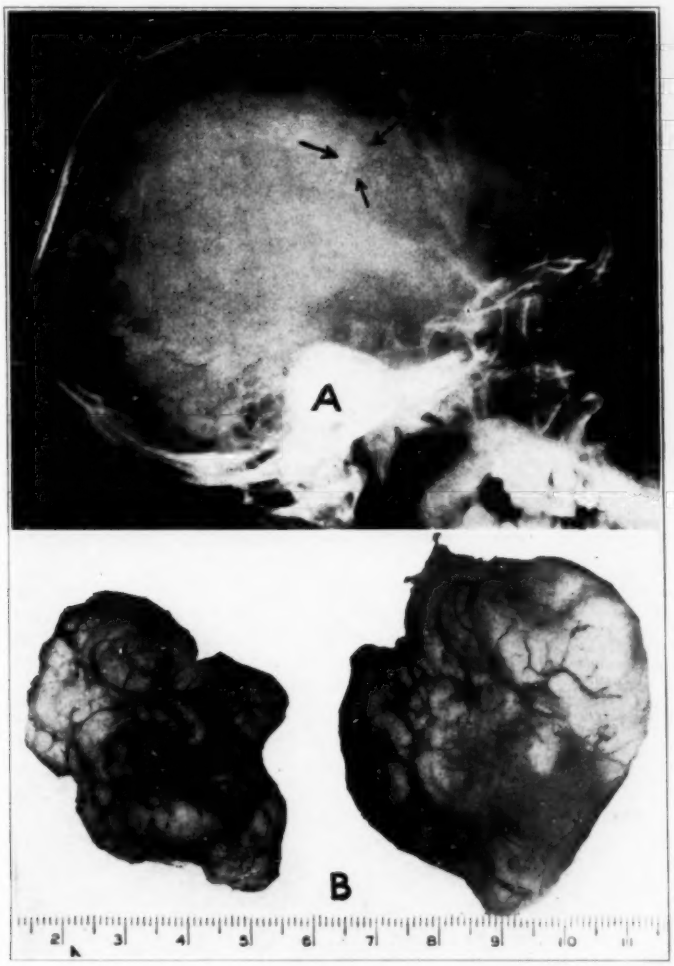

Fig. 11 (case 6).—*A*, calcium deposits localized the tumor. Grossly, the tumor consisted of two masses of yellowish-red, nodular, moderately firm tissue containing many calcareous deposits. Weight 85 Gm. *B*, two portions of tumor removed at operation.

Operation.—On March 23, 1928, Dr. Sachs turned down a unilateral occipital flap; just beneath the thinned cortex of the left occipital lobe the edge of a circumscribed mass was seen, which on further examination was found to

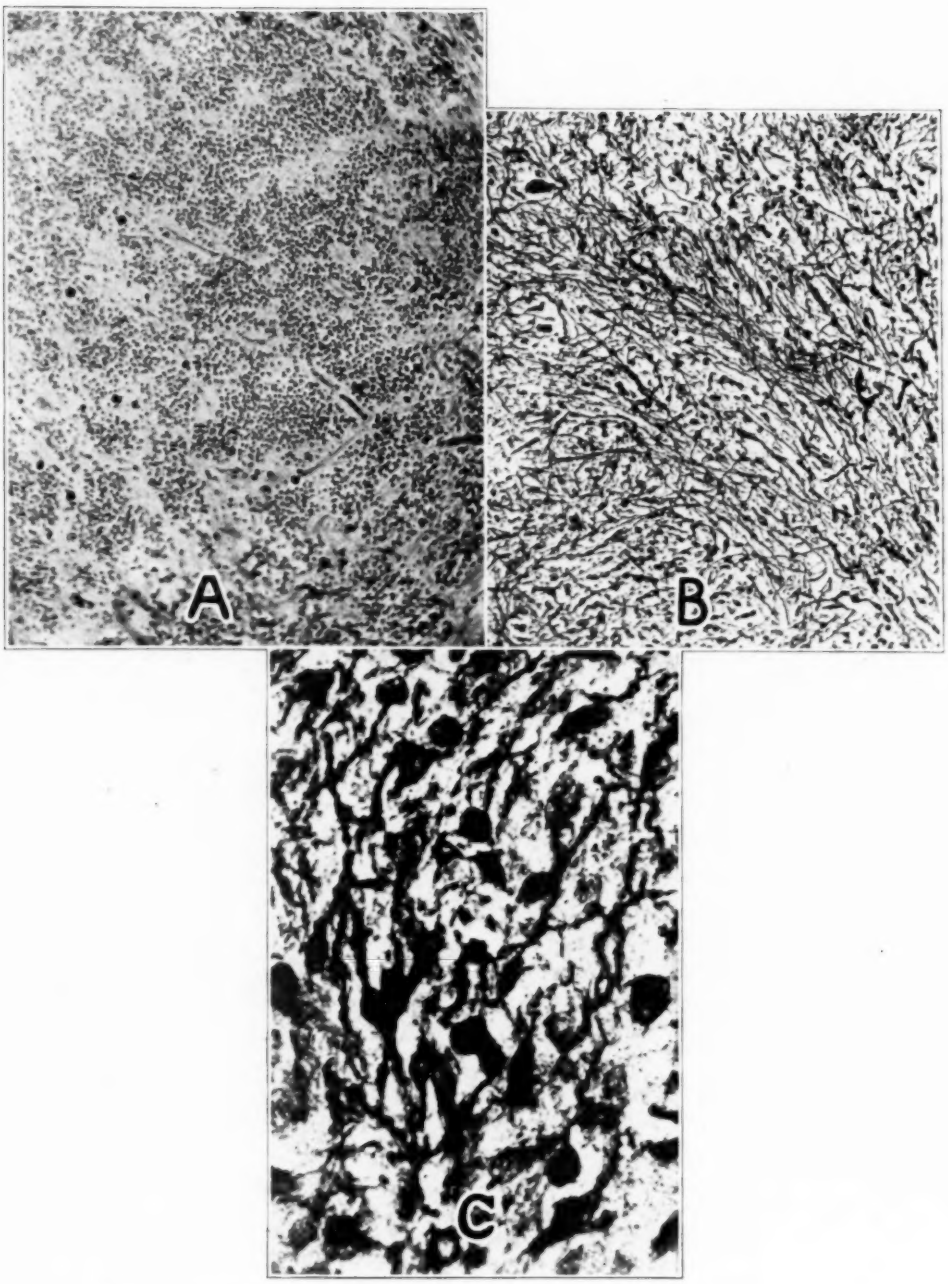

Fig. 12 (case 6).—*A*, the cells are seen to be closely packed as if in compartments formed by the blood vessels. Hematoxylin and eosin stain; × 80. *B*, illustrating the length and arrangement of the processes of the ependymal spongioblasts. Silver carbonate stain; × 88. *C*, high power of *B*. Note that the cells are bipolar as well as unipolar. Silver carbonate stain; × 580.

extend down to the tentorium into the region of the cuneus. Two large cysts were encountered on the surface of the tumor, which when evacuated permitted further dissection of the tumor; the latter was attached to the posterior horn of the lateral ventricle. The ventricle gaped widely following the enucleation of the growth. The immediate postoperative rise of temperature returned to almost normal on the following day, but every third or fourth day thereafter for a month there was

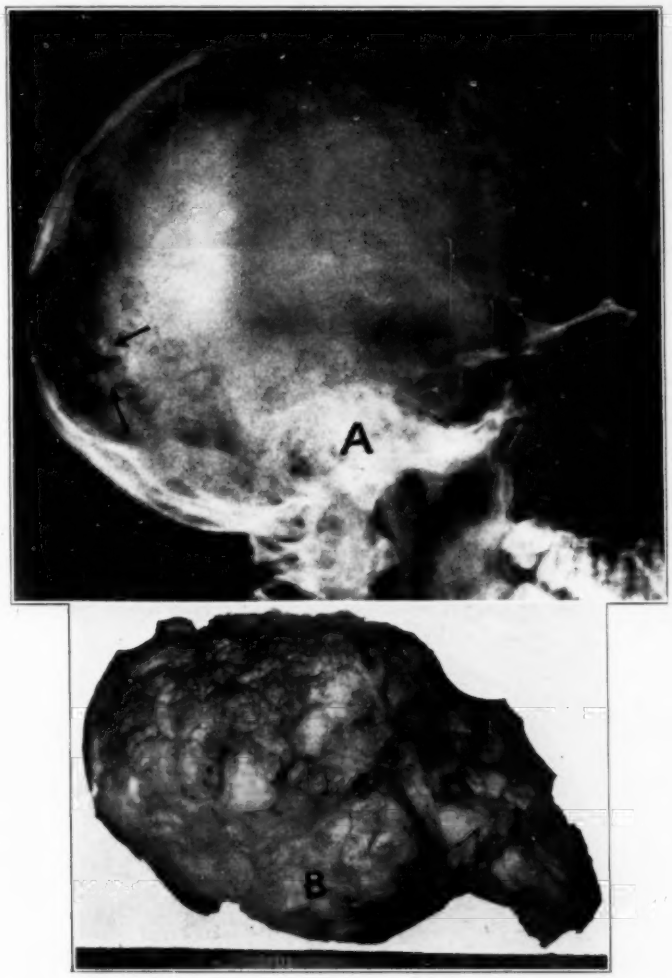

Fig. 13 (case 7).—*A*, calcified nodules in the occipital region and the field defect prompted an exploration of the occipital area. *B*, there were several large cysts on the surface of this tumor which were ruptured at operation.

a sudden rise to over 40 C. (104 F.), the temperature falling almost as abruptly as it had risen following the withdrawal of an ounce or two of cerebrospinal fluid by lumbar puncture. On May 6, 1923, the patient was discharged in excellent condition, save that the little sight in the right eye had disappeared.

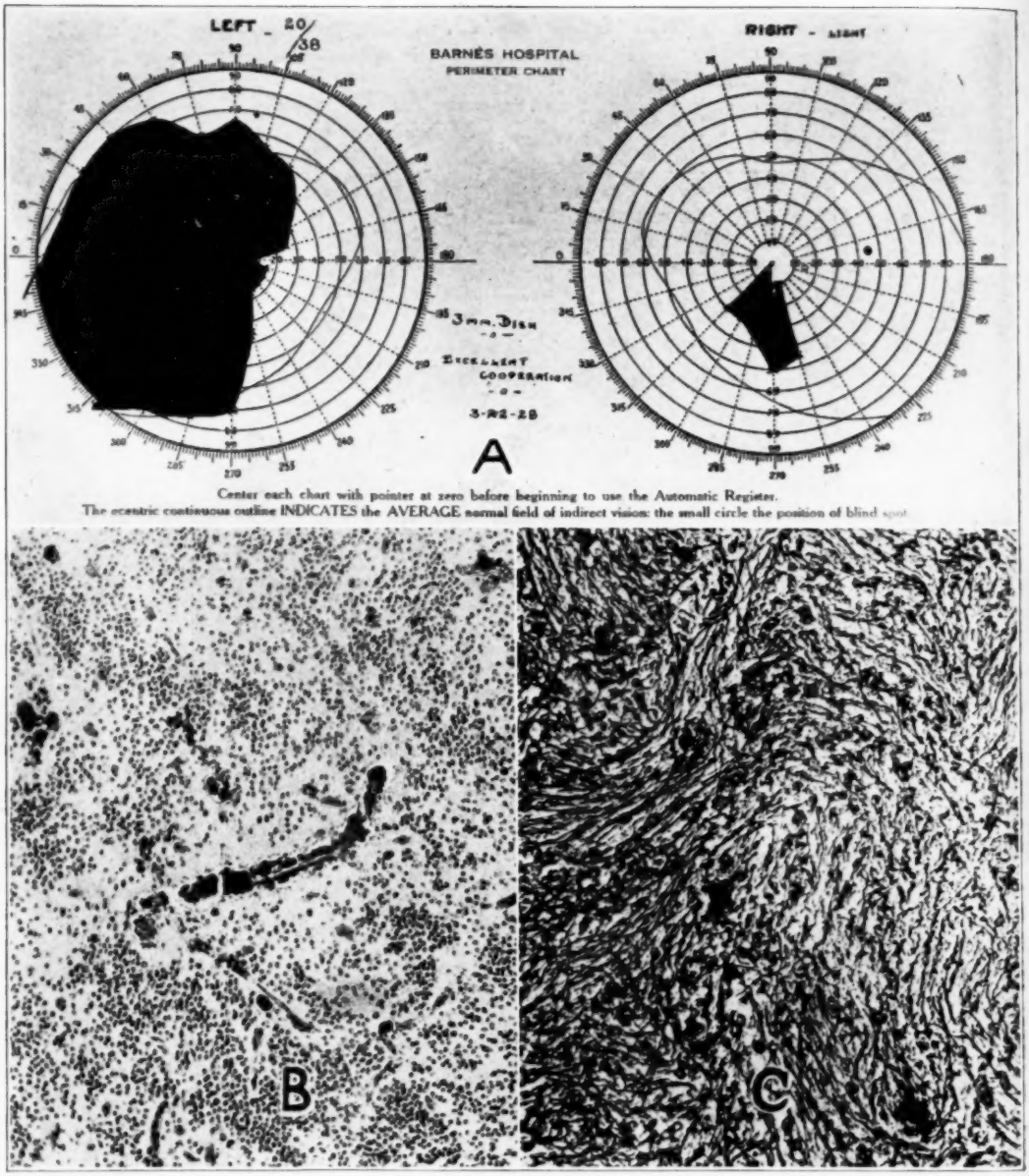

Fig. 14 (case 7).—*A*, visual fields in case 7. *B*, calcium deposits are found scattered about in the tissue and in the walls of the blood vessels. Hematoxylin and eosin stain; × 88. *C*, illustrating in a most striking way the long processes of the ependymal spongioblasts. Such a microscopic picture seems to be characteristic as no other glioma stained with silver carbonate has in our experience presented such a picture. Silver carbonate stain; × 88.

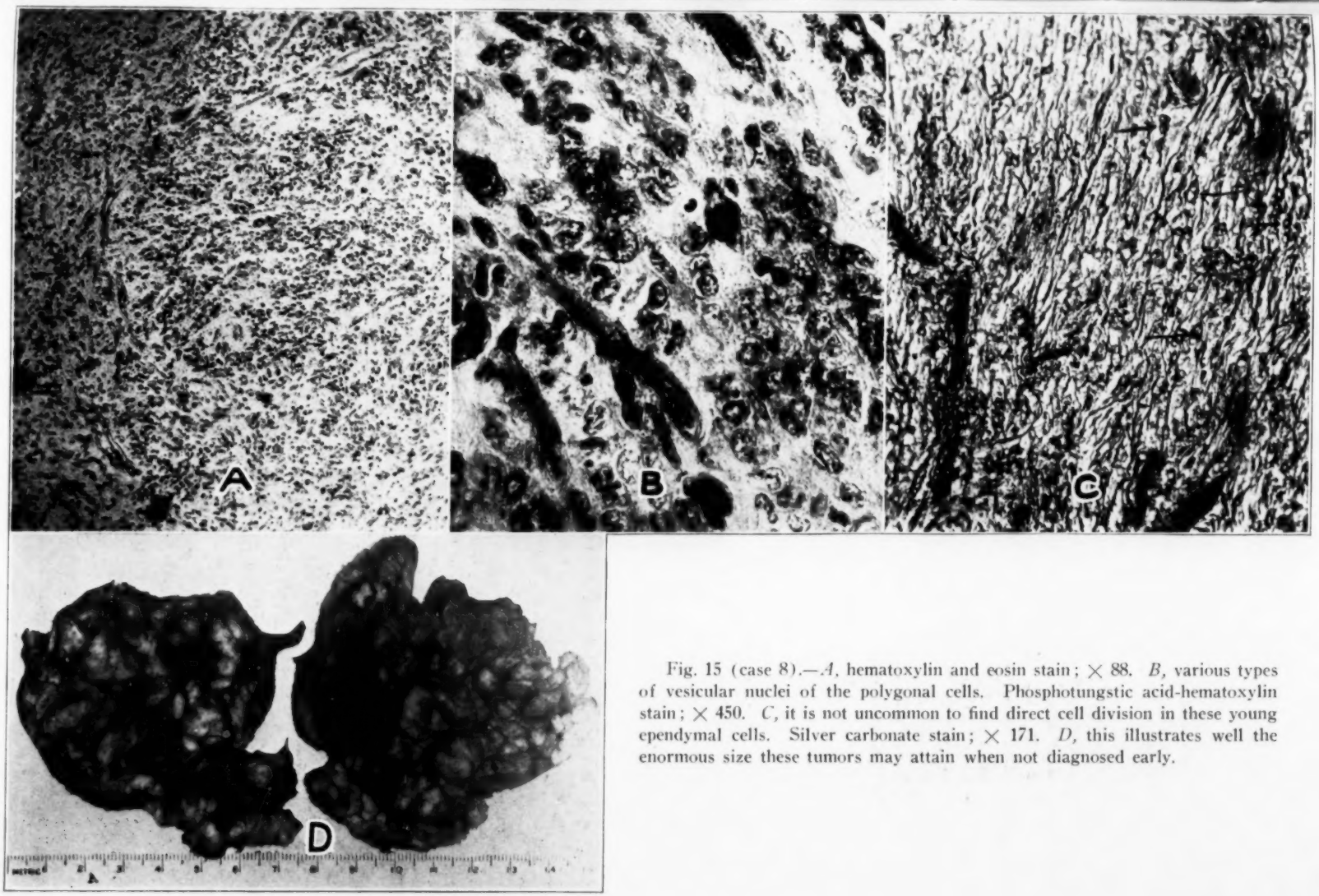

Fig. 15 (case 8).—*A*, hematoxylin and eosin stain; × 88. *B*, various types of vesicular nuclei of the polygonal cells. Phosphotungstic acid-hematoxylin stain; × 450. *C*, it is not uncommon to find direct cell division in these young ependymal cells. Silver carbonate stain; × 171. *D*, this illustrates well the enormous size these tumors may attain when not diagnosed early.

The solid portion of the tumor weighed 86 Gm. It was definitely encapsulated, nodular and moderately firm, and one or two cysts were seen on the surface. On section it was vascular, and was a mottled pinkish gray.

Histology.—Microscopically, the predominating cell was the ependymal spongioblast. As seen in sections stained with hematoxylin and eosin, these cells were closely packed, at times simulating an acinous-like formation, but most frequently looking as if packed in compartments formed by the numerous blood vessels. The clear zones between the cells and vessel walls were everywhere distinctly seen. Sections stained with phosphotungstic acid showed that these clear spaces were filled with small fibers that came from the large vesicular cells. In the large polygonal cells, blepharoplasts could be seen under high magnification. The silver carbonate impregnations gave a distinctive and characteristic appearance, the long heavy tails streamed from one or both ends of the cells, paralleling each other in many places, and in other areas intermingling in such a manner that the cell bodies were almost hidden.

CASE 8.—*Ependymoma of the Right Motor Region.*

W. W., a boy, aged 10, entered the Children's Hospital on Aug. 15, 1928, with a history of right-sided headaches which began in July, 1927. These had been accompanied by vomiting. A left-sided paralysis began in April, 1928. On examination there was found a left hemiplegia, with left facial weakness and bilateral choking of the optic disks; the reflexes on the right were hyperactive, and the left abdominal reflex was absent. X-ray pictures of the skull showed a marked separation of the frontal sutures.

Operation.—On Aug. 20, 1928, Dr. Klemme turned down a right parietal bone flap. When the dura was opened, a rather firm, sharply demarcated, red, nodular tumor was seen lying in the motor region of the brain. When the tumor was dissected, it was found to be attached to the inside of the right ventricle, and it filled the entire temporoparietal region. It was completely removed. The patient collapsed, however, and died before leaving the operating table.

Autopsy.—Grossly, the tumor consisted of two large masses weighing together 244 Gm. It was red, rather soft and very nodular, and looked not unlike a cluster of grapes. No cysts were seen. Section of the tumor showed a tough central fibrous core with a few small degenerated areas in some of the lobulations.

Microscopically, the tissue was cellular and vascular channels were found throughout the section. The cells with their vesicular nuclei were closely packed; some were oval, others were spherical, and they contained variable amounts of chromatin material. No mitotic figures were seen. In the sections stained with phosphotungstic acid, the cell processes filled the region adjacent to the walls of the blood vessels. Silver carbonate stains showed an abundance of ependymal spongioblasts the wirelike tails of which ran for long distances through the sections. Cajal's gold chloride preparations showed an occasional astroblast throughout the tumor.

COMMENT

It is noteworthy that the majority of ependymomas in this collection were situated in the cerebrum close to the lateral ventricles, whereas this type of glioma is most commonly found in the posterior fossa. Whether a study of a large number of these growths will show that their location is as common in the cerebrum as in the posterior fossa, only the future can tell. No particular portion of the lining of the lateral

TABLE I.—*Summary of Important Clinical Observations*

Case	Age	Sex	Onset of Symptoms	Admission	Operation	Location	Results
1	17 years	M	August, 1916	December, 1916	1. Jan. 4, 1917, right subtemporal decompression	.....	No improvement
					2. Jan. 17, 1917, left subtemporal decompression	Lateral ventricles	Died one week later
2	16 months	M	July, 1917	October, 1917	1. Nov. 3, 1917, cerebellar craniotomy; decompression	Fourth ventricle	Improved
					2. May 16, 1918, cerebellar craniotomy; partial removal of tumor	Fourth ventricle	Died second day; postoperative hyperthermia
3	7 years	M	March, 1921	April, 1921	1. April 8, 1921, trephine for cerebellar abscess; left subtemporal decompression	.....	No improvement
					2. April 15, 1921, right subtemporal decompression	.....	No improvement
					3. May 2, 1921, cerebellar craniotomy; tumor seen, no attempt at removal	Fourth ventricle	Pressure symptoms subsided
					4. Sept. 9, 1921, cerebellar craniotomy; partial removal	Fourth ventricle	Died Oct. 6, 1921, from bronchopneumonia
4	9 years	F	July, 1921	February, 1922	1. Feb. 15, 1922, right cerebral craniotomy; evacuation of cyst	Right parietofrontal region	Shock
					2. Feb. 20, 1922, right cerebral craniotomy; partial removal of tumor	Right parietofrontal region	Improved
					3. Jan. 5, 1923, right cerebral craniotomy; complete removal	Right parietofrontal region	Died on operating table
5	34 years	F	June, 1926	October, 1927	1. Nov. 2, 1927, ventricle puncture; air injection		
					2. Nov. 4, 1927, cerebellar craniotomy; decompression	Fourth ventricle	Died Jan. 1, 1928, from meningitis
6	11 years	F	November, 1927	January, 1928	1. Jan. 30, 1928, left cerebral craniotomy; removal of tumor	Left frontoparietal region	Free from symptoms
7	16 years	M	October, 1927	March, 1928	1. March 23, 1928, left cerebral craniotomy; removal of tumor	Left occipital region	Free from symptoms
8	10 years	M	July, 1927	August, 1928	1. Aug. 20, 1928, right cerebral craniotomy; removal of tumor	Right motor region	Died on operating table

ventricle was a favorite site of origin for these cerebral tumors. They were encountered in various regions of the cortex. One ependymal glioma arising from the posterior horn grew out into the left occipital lobe (case 7). Another lay in the right frontoparietal region (case 4); a third was embedded in the left motor cortex (case 6). In the most recent case, the tumor was attached to the roof of the right lateral ventricle and invaded the temporoparietal area of the brain (case 8). In two of the tumors, the one in the frontoparietal region (case 4) and the one in the left motor cortex (case 6), there was no demonstrable ventricular attachment at the time of the operation; when the former specimen was studied at autopsy, careful examination revealed no attachment of the tumor to the lateral ventricular wall. These two tumors might well support the theory of embryonic cell rests as a plausible explanation of their origin. Such "cell rests" might, however, have been a congenital malformation in the infolding of the neural tube.

TABLE 2.—*Cerebral Ependymomas*

Case	Age, Years	Duration of Symptoms Before Admission	X-Ray Examination of Localizing Value	Duration of Life Following Operation
1	17	Five months	None	One week
4	9	Eight months	Calcium deposits	Eleven months
6	11	Three months	Calcium deposits	Eight months plus
7	16	Six months	Calcium deposits	Seven months plus
8	10	Thirteen months	None	Died on operating table
Average	12.6	Seven months	60 per cent	Five months plus

Of the tumors which lay beneath the tentorium, two arose from the posterior medullary velum and a third grew upward from the rhomboid fossa, being so deeply hidden that it was not seen at the operation.

Grossly, all the tumors in the cerebrum had several points in common. Each was reddish gray, definitely encapsulated, lobulated and moderately firm, and cysts, when present, were found only on the surface. The lobulations varied considerably in size and shape. The lobules in case 8 were so small but so distinctly formed that on removal the neoplasm resembled a large bunch of grapes, whereas in the other tumors the lobulations tended to be much larger. Cysts, varying in size from 1 to 4 cm. in diameter were found on the surface of the majority of these tumors. The fluid in them was a thin yellow liquid. In cases 4 and 7, in which sufficient fluid for gross examination was obtained at the operation, it did not coagulate on exposure to the air. In this respect the fluid differs strikingly from that found in other gliomatous cysts. If this point is confirmed in a larger group of cases, it may prove a valuable differentiating point at the operating table. The cut surface of these gliomas was mottled red, brown and gray, and in two of the cerebral growths calcareous deposits were of sufficient size to be seen on inspection.

The tumors in the midcerebellar region possessed similar characteristics, except that they were a little softer and showed less tendency to cyst formation and calcareous alteration.

The types of tumors discussed presented a characteristic microscopic picture, by far the majority of them falling into that group that Bailey at one time called ependymoblastomas. Sections stained with hematoxylin and eosin all showed closely packed tumor cells with large, spherical or oval, vesicular nuclei. Scattered among the cells were numerous thin-walled vascular channels made particularly conspicuous by clear areas about them. Sections stained with methods for demonstrating neurofibrils showed that these "clear spaces" were filled with fibers that ran toward the walls of the blood vessels. These were the processes of the embryonic cells. In our experience, Hortega's silver carbonate stain for demonstrating oligodendroglia has been valuable for impregnating the ependymal spongioblasts. When stained by this method, the tadpole-shaped embryonic cells presented a characteristic appearance with their single long heavy, wavy tails. These processes were much larger and coarser than the processes of their antecedents, the primitive spongioblasts. They could be traced for a considerable distance in the sections. These ependymoblastic processes at times formed tangled masses of fibers, or in places definite parallel rows which gave a picture of a whorl-like formation. These ependymal spongioblasts were so distinctive when stained with Hortega's silver carbonate methods that their presence in the sections seemed to us as great a diagnostic aid in classifying these tumors as the presence of the polygonal ependymal cell and the observation of blepharoplasts. In nearly every histologic study, calcified plaques were found scattered among the cells or along the walls of the blood vessels. For some reason that is not clear to us the supratentorial tumors showed much more calcareous deposit than the tumors in the region of the fourth ventricle.

In three of the five cerebral ependymomas the clusters of circumscribed calcareous deposits were of sufficient size and density to show distinctly in x-ray pictures of the skull, aiding materially in localizing the lesion. Such aggregations of calcareous nodules in films of the skulls of children seem to be distinctive. In the experience of this clinic, no other type of glioma occurring in childhood has shown calcium deposition, so that we have recently ventured to predict that an ependymoma would be found on the basis of this x-ray observation.

The presence of an ependymal tumor in a woman, aged 34, raises the average age to 13 years. The average lapse of time between the onset of symptoms and admission to hospital was five months; if the adult is omitted from this average, the period was a trifle over three months. Whether all these tumors have lain dormant for a long period of time is uncertain, for once the symptoms appeared, the tumors seem

uniformly to have grown rapidly. In cases 3 and 4, in which the tumor was exposed in the posterior fossa and cerebellar decompression was resorted to without disturbing the tumors, the patients did poorly and were in extremis in less than two months. In case 4, in which partial removal of a cerebral tumor was accomplished, the remaining neoplasm seemed to grow more rapidly; roentgen therapy apparently did not check the growth, and at the second operation, eleven months later, the tumor was much larger than the one removed at the first operation. In case 2, in which radiation was given with radium emanation needles, no evidence of any beneficial results was noted.

In cases 6 and 7, in which we believe a complete removal of the tumors was accomplished, no return of symptoms has been noted after a lapse of fourteen and thirteen months, respectively. This time is too short to form any final conclusions, but one might expect an excellent prognosis when such well encapsulated tumors of the cerebrum have been removed in toto. The size which these tumors may attain before coming to operation is illustrated in cases 4 and 8; this always makes the surgical procedure formidable. The ependymomas of the cerebrum offer a favorable prognosis, for they are more accessible than this type of tumor when it arises in the posterior fossa. With the improved technical methods of the last few years, it is believed that even the tumors of the posterior fossa can be handled more successfully.

SUMMARY

1. Eight cases of tumor of ependymal origin are reported, of which five occurred in the cerebral hemispheres.

2. Ependymomas in the cerebral hemispheres, being encapsulated tumors, offer a favorable prognosis.

3. Calcareous deposits in roentgenograms of the skull were of definite localizing value in three of the supratentorial lesions. In the experience of this clinic, ependymomas have been the only type of gliomas showing such markings in children.

4. The observation of cystic fluid which did not coagulate may be of differential diagnostic value at the operating table.

5. Ependymal spongioblasts as demonstrated with Hortega's silver carbonate methods have been the most valuable histologic aid in establishing a diagnosis of these tumors.

PYRAMIDOPALLIDAL DEGENERATION SYNDROME DUE TO MULTIPLE SCLEROSIS

A CASE PRESENTING SPASTIC PARAPLEGIA BELOW THE WAIST AND PARALYSIS AGITANS AND ASTEREOGNOSIS ABOVE *

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Aside from their occurrence in cases of encephalitis, combined clinical syndromes of the pyramidal and extrapyramidal systems are rare. Lhermitte, Cornil and Quesnel [1] described a syndrome based on a case in which the symptoms of pseudobulbar palsy were associated with extrapyramidal rigidity. Claude and Alajouanine [2] reported a case that was similar, but in which Babinski reflexes could not be elicited, while the lower extremities were too rigid to permit walking. Lhermitte and McAlpine [3] reviewed these cases and the syndrome of Lhermitte, Cornil and Quesnel and presented a somewhat similar case of what they termed a new syndrome that differed from the foregoing in several apparently essential features, notably in the absence of the syndrome of pseudobulbar palsy and in the presence of an additional symptom—choreiform movements. Jakob [4] stated that, in cases of cerebral

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* From the Battle Creek Sanitarium, the Clifton Springs Sanitarium and the Michigan State Psychopathic Hospital.

1. Lhermitte, J.; Cornil, L., and Quesnel: Le syndrome de la dégénération pyramido-pallidale progressive, Rev. neurol. **27**:262 (March) 1920.

2. Claude, H., and Alajouanine, Thomas: Hypertonie généralisée avec troubles d'aspect pseudobulbaire, rire et pleurer spasmodique, chez un adulte. Localization pallidale probable, Rev. neurol. **29**:657 (May) 1922.

3. Lhermitte, J., and McAlpine, D.: A Clinical and Pathological Résumé of Combined Disease of the Pyramidal and Extrapyramidal Systems, with Especial Reference to a New Syndrome, Brain **49**:157 (June) 1926.

4. Jakob, A.: The Anatomy, Clinical Syndromes and Physiology of the Extrapyramidal System, Arch. Neurol. & Psychiat. **13**:596 (May) 1925.

arteriosclerosis, he frequently had seen combinations of partial pyramidal and extrapyramidal lesions. In our case, the freakish characteristics of a pure spastic paraplegia were manifested below the waist, and paralysis agitans and astereognosis above, the symptoms having appeared, in turn, first on the right, then on the left.

REPORT OF CASE

History.—A physician, aged 73, formerly of considerable ability, had spent the last forty years in China, except for vacations. There, prior to the onset of the disease here described, he had had malaria and relapsing fever. In 1881, at the age of 26, he had begun to lose the hearing in the right ear, owing, he said, to a catarrhal condition in that ear. During the course of this trouble he had developed attacks of Ménière's syndrome; he remembered having fallen off his chair in an attack. The attacks of vertigo had soon ceased, but the hearing had remained poor.

The first indication of the disease here discussed was in 1905, when at the age of 50, he began to suffer from sensory disturbances in the left leg, cold seats feeling warm but hot objects, hot. This spread to the opposite side and continued for a year or two. Then his attention was attracted to peculiarities in the sense of smell. The perception of odors would persist for hours, and odors were often perceived where none existed. Shortly after this, he noticed a slight weakness in the legs and a stinging sensation around the anus.

By 1912, he had difficulty in walking, owing to spasticity of the right leg. This was particularly noticeable when he was tired or constipated. He also noticed dimness of vision and a blind spot in the field of the left eye.

In May, 1912, an examination at the Battle Creek Sanitarium by Dr. M. A. Mortensen gave the following relevant data: spastic gait, more in the right leg; increased patellar reflexes, more on the right; positive Babinski reflex and ankle clonus on the right only; increase of both achilles reflexes; active, but not abnormal deep reflexes in the arms; absence of abdominal reflexes; diminution of the cremasteric reflexes. All sensation was normal, but a "suggestion of nystagmus" was present.

In August, 1912, there was a distinct band of hyperesthesia and girdle sensation running from the center of the spine toward the left, ending in the midline anteriorly and extending from the eighth to the tenth dorsal segments. A roentgenogram of the spine did not show any pathologic changes, except that the twelfth rib on the left was congenitally absent.

A chart of the fields of vision showed a central scotoma for color in both eyes and also for white in the left. In the right eye there was another scotoma for green and red. Both fields of vision were contracted for white, red and green.

In September, 1912, ankle clonus was not obtainable on the right, and the band of hyperesthesia had disappeared. It should be noted that the band of hyperesthesia and girdle sensation were present for only one month. (They never returned.) After this examination, the patient returned to China, and there he definitely improved, so that he walked fairly well for about two years.

In 1916 and 1917, he developed urinary irregularities, especially in the form of urgency, and this condition forced confinement of his activities to office work. The difficulty with gait soon made it impossible for him to be about much. The parosmia continued.

By 1921, the legs were so spastic that he could not walk without assistance, and the right hand had begun to shake during excitement. Soon, the left hand began also to shake, while the shaking on the right increased. By 1923, he could walk by pushing a wheel chair, but not otherwise, and the right hand had developed such uncertainty that he could no longer sign checks. This difficulty was in the nature of sudden, unexpected, involuntary movements. He trained the left hand to sign his name. The bladder had become automatic, i. e., it would fill to a certain point and then discharge involuntarily. There never were any tremors, or choreic or athetoid movements of the lower limbs.

In 1927, the patient, completely incapacitated for work, returned for another examination.

Examination.—There was not any lack of expression, and not any sialorrhea. Involuntary laughing or crying was not noted, but a slight emotional exaggeration like that in paralysis agitans was seen at times. The intellectual faculties were preserved.

Pseudobulbar symptoms were absent.

Voluntary motor power in the face and neck was normal; the arms and hands were weak and slow of movement; the legs were practically paralyzed. The head was held well up. There was an upper dorsal kyphosis, with scoliosis of the entire spine to the right (from lying constantly on the right side). The right shoulder was elevated; the arms were abducted; the hands were typical of paralysis agitans. The thighs were flexed, the legs adducted, the knees semiflexed and the ankles extended. Occasional mild choreiform movements were noted in the right arm and hand. Constant tremor during rest was present in the head and in the small muscles of both hands and the rotators of the forearms. The tremor was temporarily under voluntary control; it was absent during sleep.

Early, the gait was spastic; later, the patient was bedridden because of spasticity and contractures. The trunk moved en masse. Muscle tone was moderately increased for days at a time in the arms and hands, especially on the right; this was entirely absent at other times. The rigidity was plastic and became lessened in a few seconds on passive motion. The legs were adducted. All the leg muscles were tense in contracture. Cogwheel phenomena were not seen. Early there had been paresthesias; later, sensation was entirely normal. Passive flexion of the knee joints caused an extensor response in the hallux. Other abnormal associated movements were not observed.

The deep arm reflexes were normal. The Klippel-Weil phenomenon was absent. The abdominal and cremasteric reflexes were absent. The knee jerks were apparently normal, but were masked by spasticity. Bilateral Babinski, Strümpell and Chaddock reflexes were present with clonus. The defense reaction was active.

In making Bárány tests a response was not secured to caloric tests on the right; there was reduced reaction on the left. Advanced bilateral nerve deafness (70 units loss in the right) was present.

Sphincters: Bladder action was automatic, and there were large trabeculations in the bladder wall. There was a lack of tone in the anal sphincter.

The pupils, 4 mm. in size, were equal and round, and reacted well to light and on convergence. Nystagmus and pseudonystagmus were not observed. There was a clearcut physiologic cup with much stippling of the lamina cribosa; the nerve itself was not atrophic but appeared senile; the arteries were sclerosed. There was a scotoma for green in the right field (Dr. Stegman).

Miscellaneous Observations: A Wassermann test of the blood was negative. The spinal fluid was under 90 mm. of water pressure, clear and colorless; the sugar content was 75 mg. per one hundred cubic centimeters; cells were not

seen; a Wassermann and a Kahn test were negative; the colloidal gold curve in a first test was 5444320000, in a second 4333441000. The arteries were sclerosed and tortuous; the blood pressure was from 120 to 150 systolic, and 80 diastolic. Parosmia persisted (lilacs smelled like vegetables). Complete astereognosis in the right hand had developed in the preceding three years; it was slight in the left, with perfect preservation of sensation of all types, camel's hair touch, pain, temperature, tactile localization and tactile discrimination (compass test), and muscle, joint and vibration senses. He described a round bottle, 8 inches long, as irregular in shape, hard, cold, smooth in general but with rough areas and as being about 5 inches long, but he did not have an idea as to what it might be. Cerebellar symptoms were absent.

Course.—In 1928, a general weakness and bulbar symptoms developed. Owing to the dysphagia, a marked inanition and later a purulent bronchitis ensued, causing the death of the patient.

Chronologic Summary of Symptoms.—The symptoms occurred in the following chronologic order: in 1905, paresthesia of buttocks, parosmia; in 1912, a brief period of paresthesia, spastic gait, scotomas; from 1914 to 1915, a remission; in 1921, increasing spastic paraplegia, beginning of parkinsonian tremor in the right hand, beginning of astereognosis; in 1927, nearly total paralysis of the lower extremities, parkinsonian tremor of the hands and head, without pyramidal lesion of the upper extremities, but with mild choreic movements and total astereognosis on the right, moderate on the left; mental faculties well retained. There never were any tremors in the lower extremities. Death occurred in 1928.

COMMENT

After a careful and exhaustive postmortem examination, Lhermitte and McAlpine summarized the pathologic observations in their case as follows: 1. A marked reduction appeared in the number of the cells of the putamen, with an accompanying neuroglial overgrowth, which, although it was not absolutely confined to this region, was more marked here than elsewhere. Degeneration was present in many of the fibers running from the putamen into the globus pallidus and ansa lenticularis; many of the fibers having origin in the caudate nucleus were also affected. 2. A slight but definite reduction in the number of the motor cells of the globus pallidus was present, with a neuroglial overgrowth, which was less than that in the putamen, especially the overgrowth of those cells that go to compose the ansa lenticularis. 3. Degeneration of the pyramidal tract was not evident above the level of the medulla. In the cord there was degeneration of the crossed pyramidal tracts.

The pathologic observations in the cases mentioned by Jakob consisted of multiple small arteriosclerotic foci in the gray or white matter of the centralis anterior, as well as similar lesions in the striopallidum and the thalamus or in the frontoparietocerebellar system.

The case reported here differs clinically from the case of Lhermitte and McAlpine in the following points: (1) the tremor of the head, chin, forearms and hands like that of paralysis agitans; (2) the uncomplicated lesion of the pyramidal tracts of the lower extremities; (3) the dis-

turbances of the sphincters; (4) the parosmia and the scotomas, and (5) the astereognosis. There was also the probably irrelevant Ménière's syndrome with its sequelae.

We considered the possibility that the case might be one of lesion of the spinal cord, the patient developing, later in life, an ordinary paralysis agitans. The symptoms of the latter could not, in that case, manifest themselves in the lower extremities. But the development of parosmia and scotomas simultaneously with the spastic paraplegia indicated simultaneous pathologic changes in the cerebrum. Further, the definite astereognosis did not, in any way, belong to paralysis agitans.

A diagnosis of multiple sclerosis was carefully considered, but rejected. Pointing to multiple sclerosis were the early paresthesias with developing spastic paraplegia; absent abdominal reflexes; transient scotomas; deafness; the diffuse distribution of the cerebral lesions; the sphincter disturbances; the remission of symptoms; the paretic gold curve, and the termination in contractures. The disease, however, began when the patient was 50 years old, and progressed, except for one long remission, for twenty-three years; each symptom, except the paresthesias, occurred first on one side, then, shortly afterward, it was duplicated on the other, and each was referable, anatomically, to the spinal cord, the lenticular nuclei and the superior parietal lobules, and yet not one classical symptom referable to the eyes, speech or tremor had appeared by the time that the patient died, at the age of 73. There is a possibility that Ménière's syndrome was also due to the multiple sclerosis. In that case, the disease began at the age of 26 and continued for forty-seven years.

REPORT OF NECROPSY

Gross Observations.—The brain appeared normal. Convolutional atrophy was not greater than one might have expected in view of the patient's age. As the cerebrum was sectioned transversely, pea-sized grayish plaques were seen in every section (figs. 1 and 2). Similar plaques were seen in transverse sections of the cord. Along the dorsum of the spinal cord, adherent to the pia mater, were found numerous osseous or cartilaginous plaques measuring from 3 to 4 mm. in diameter, which were roughly quadrangular in shape and from 1 to 2 mm. in thickness.

Microscopic Observations.—Sections from the cortex showed mild fibrosis of the pia mater with recent hemorrhage. The surface of the convolutions was markedly irregular and, with an increase in the marginal feltwork, there was a definite indication of cortical atrophy. The cyto-architectonic arrangement was not essentially disturbed in any of the areas examined. There were many small corpora amylacea in the surface of the marginal layer. The blood vessels of the cortex showed a definite increase in fibrous tissue. In general, the nerve cells showed a considerable amount of fatty pigment, in keeping with the age of the patient. The cells were fairly well preserved.

Sections of the optic tracts showed irregular degeneration, especially on the right side.

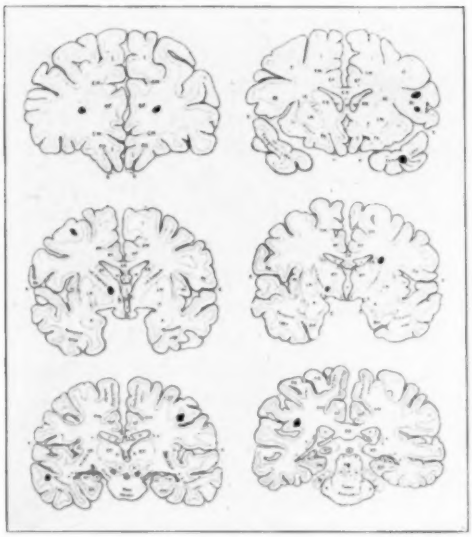

Fig. 1.—Diagrammatic representation of the lesions of the brain as seen in cross-sections in a case of multiple sclerosis presenting the pyramidopallidal degeneration syndrome.

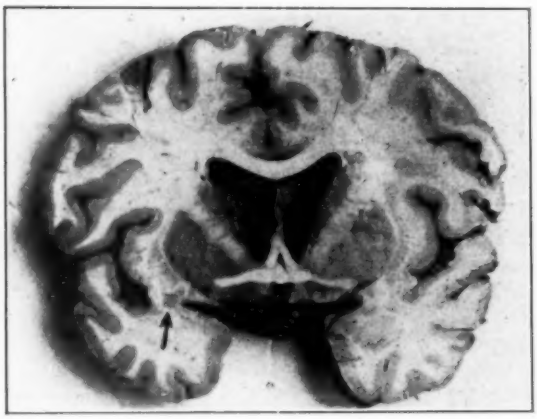

Fig. 2.—A transverse section of the brain in a case of multiple sclerosis presenting the pyramidopallidal syndrome.

Fig. 3.—A transverse section of a cervical segment of the spinal cord, the Kulschitzky stain showing myelin defects, nonsystemic in character; × 5.

Fig. 4.—A transverse section of the spinal cord in the midthoracic region, the Kulschitzky stain showing a wedge-shaped defect and the resulting symmetrical atrophy; × 5.

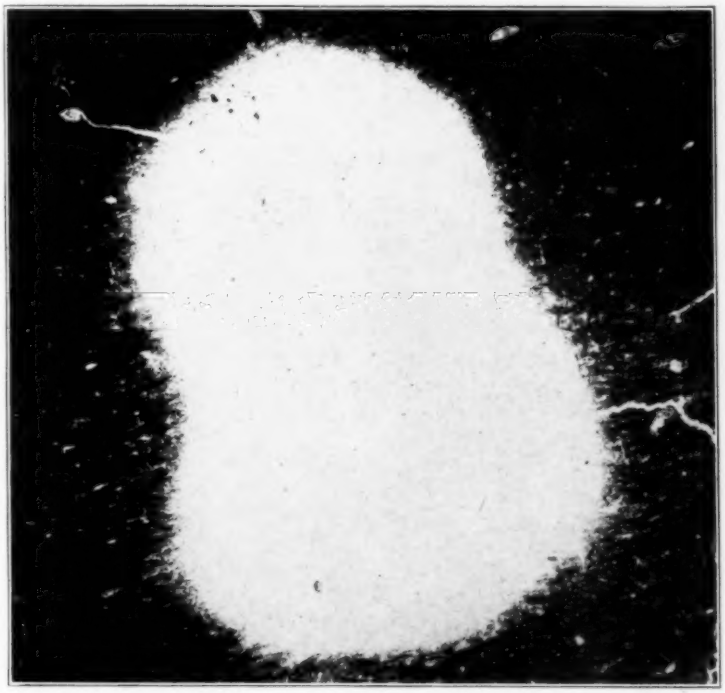

Fig. 5.—A myelin defect in the white substance of the frontal lobe, showing a characteristic loss of myelin in a sharply circumscribed plaque (Spielmeyer stain); × 17.

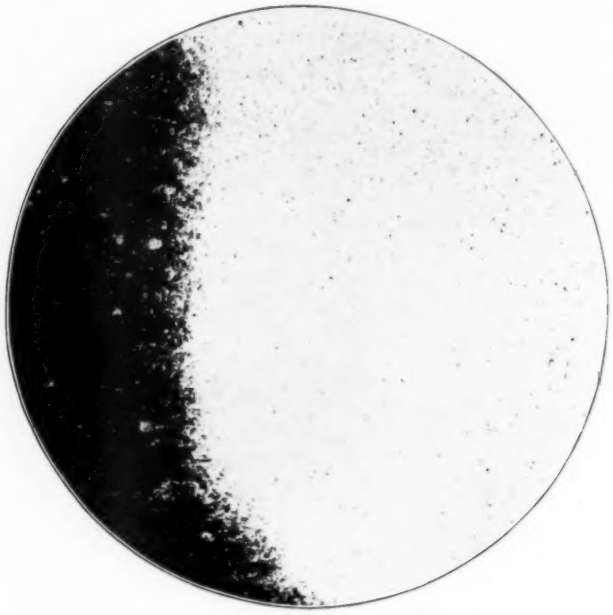

Fig. 6.—A portion of the section of the spinal cord shown in figure 3, taken at the border of the plaque. It shows normal myelin sheaths, and, in the area of the defect, faintly stained neuroglia; × 625.

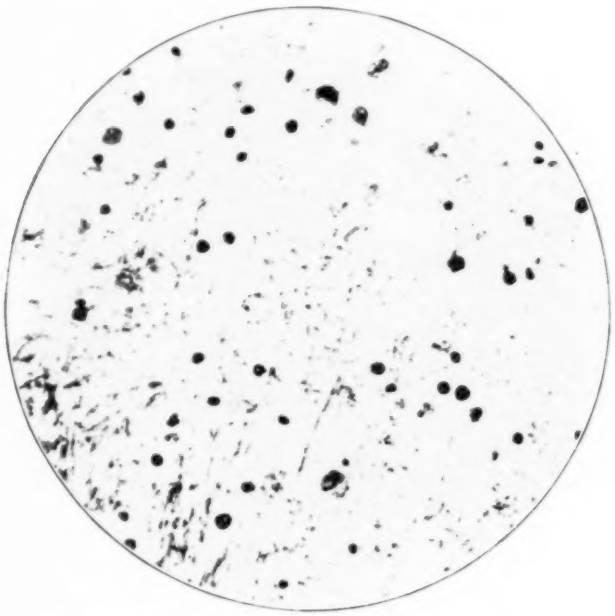

Fig. 7.—A Bielschowsky preparation revealing the persistence of the axis-cylinders into the sclerotic plaque; × 500.

Sections of the spinal cord and bulb were interesting. Plaques were not seen in the brain stem, but below the medulla they were numerous. Degeneration was found in the marginal tracts at the first and second cervical segments, in three other zones in the third and fourth cervical segments, in the posterior columns in the fifth cervical segment, in the anterior motor and in one posterior cerebellar tract in the sixth cervical segment, and in similar irregular areas in the lower segments. At the sixth dorsal segment, a large plaque was found involving the crossed pyramidal tracts, the posterior horn and the fasciculus cuneatus all in one.

There was, therefore, no secondary degeneration.

Recent petechial hemorrhages were found in the optic tracts and throughout the cord.

Diagnosis.—The pathologic diagnosis is obviously multiple sclerosis.

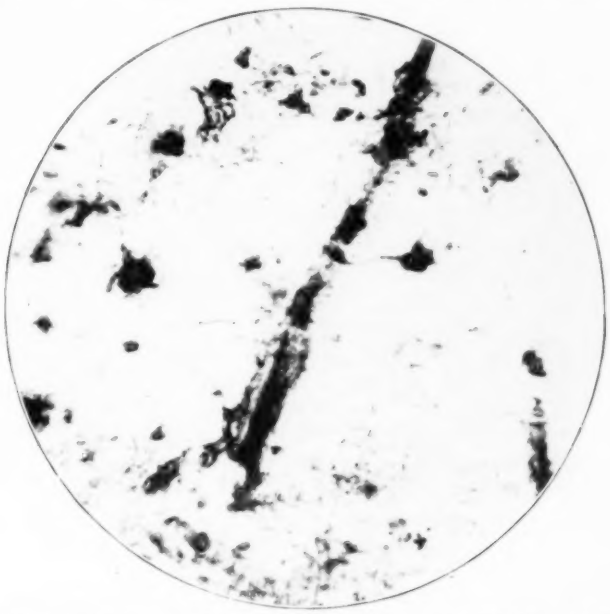

Fig. 8.—Fibroglia in the plaque shown in figure 3 (Cajal's gold sublimate); × 500.

SUMMARY

1. Prior to this report three pathologic entities were known to cause clinical pictures of combined pyramidal and extrapyramidal syndromes: epidemic encephalitis, degeneration in the basal ganglia and crossed pyramidal tracts of the cord, and cerebral arteriosclerosis. A fourth is added: multiple sclerosis.

2. An extremely unusual case of multiple sclerosis is presented, manifesting spastic paraplegia below the waist, and a paralysis agitans syndrome and astereognosis above. The disease began when the patient was 50 years old, and continued for twenty-three years. It ended in death without the appearance of any of the classical symptoms referable to the eyes, speech or tremor.

ABSTRACT OF DISCUSSION

DR. THOMAS J. HELDT, Detroit: More superficially observed cases of this kind are probably usually grouped clinically as multiple sclerosis. Only through detailed studies such as those made by Dr. Nielson do we come to appreciate the finer differentiations.

DR. WALTER FREEMAN, Washington, D. C.: This case is interesting from several standpoints. 1. The irregular involuntary movements in association with a plaque occupying the position of the corpus Luysii. Some recent work on hemichorea seems to show that this syndrome is due to involvement of this body. 2. The pathologic picture reminds one considerably of multiple sclerosis. The clinical picture of the parkinsonian tremor suggests paralysis agitans. Is it possible that we have a combination of these two diseases? Some recent investigations indicate a bacterial origin for both these diseases, the organisms appearing much the same. Some years ago, Dr. Spiller reported the occurrence of a definite attack of encephalitis with a histologic picture indistinguishable from multiple sclerosis. May we not look on this patient as having an infection of long duration producing both the clinical picture of paralysis agitans and the pathologic picture of multiple sclerosis? I should also like to ask Dr. Nielsen whether the substantia nigra was investigated histologically. It seems to me that this is the location of the severest changes in the parkinsonian syndrome.

DR. J. M. NIELSEN, Battle Creek, Mich.: In answer to Dr. Freeman, we found no lesion whatever in the substantia nigra. Of course, the final slides are not out, and we may find something by the silver methods that we did not find by the other. The cells were intact throughout. Lhermitte and McAlpine found a definite reduction in the number of the cells of the putamen, also a slight reduction in the number of the cells of the globus pallidus and a marked destruction of the fibers composing the ansa lenticularis. We did not find anything of that kind. They also found a degeneration in the lateral columns of the spinal cord. As to the association of the two diseases, of course, it is to be considered. We have thought about it. If that is the case, the definite astereognosis should be due to the multiple sclerosis. But it appeared with the paralysis agitans, chorea and astereognosis, and all this came on after the spastic paraplegia.

OPTIC NYSTAGMUS

II. VARIATIONS IN NYSTAGMOGRAPHIC RECORDS OF EYE MOVEMENT *

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Normal optic nystagmus may be defined as that conjugate response of the eyes to a succession of moving stimuli, which is composed of an alternation of slow and quick phases in opposite directions, when each slow phase consists of the pursuit or fixation of a moving object; each quick phase represents the fixation of a new object of pursuit within the moving field. The slow or pursuit phases consequently consist of eye movements regularly in the direction of the moving visual field. The quick phases of refixation are usually in the opposite direction, determined by some peripherally seen object which at the instant engages the attention or automatically becomes the new object of regard.

This phenomenon has been studied for the most part by direct observation of the response of the eyes to a moving visual field of relative simplicity in the form of alternate vertical black and white stripes on a revolving cylinder. Direct observation, however, is incapable of analyzing the complex phenomenon. Fox and Holmes [1] frequently noted responses of the eyes which were impossible to describe by unaided observation, and they were compelled to limit their published results to those cases in which the optic nystagmus was apparently normal in one direction of rotation of the field and absent or grossly disturbed in the other. There seems little doubt that some form of graphic recording device is essential for the analytic and comparative study of the finer details of all types of eye movements. This proved true for the behavior of the eyes in reading and in vestibular nystagmus.

Several forms of eye-movement recording are now available to the student of optic nystagmus. In particular, Ohm [2] has devoted himself to

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* From the School of Medicine, Department of Internal Medicine, the Institute of Psychology, Yale University and the Medical Service of the New Haven Hospital.

1. Fox, J. C., and Holmes, G.: Optic Nystagmus and Its Value in the Localization of Cerebral Lesions, Brain **49**:333 (Sept.) 1926.

2. Ohm, J.: Das Augenzittern als Gehirnstrahlung, Berlin and Wien, Urban & Schwarzenberg, 1925; Zur Theorie des optischen Drehnystagmus. Eine Antwort an Cords, Klin. Monatsbl. f. Augenh. **78**:218 (Feb. 28) 1927.; Zur Augenzitternkunde. VIII. Der optische Drehnystagmus bei verschiedenen Gehirnkrankheiten, Arch. f. Ophth. **118**:529, 1927.

mechanical graphic technic and has published extensive kymographic records of normal optic nystagmus and its pathologic alterations. These records were obtained by means of a lever resting tangentially on the eyeball. Cords [3] has severely criticized Ohm's technic, pointing out that false movements or instrumental artefacts are produced by the recording system, and claiming that these had been erroneously interpreted by Ohm as real eye movements. There is no doubt, however, that Ohm has collected much valuable information by this method, especially concerning the gross changes in the eye movements under various pathologic states. Ohm's speculations regarding the physiology of the mechanism and its various disorders have naturally been largely brought into question by Cords' criticisms. Finer and more reliable technics seem to be a real desideratum, especially those that do not complicate the records by the interposition of long recording levers.

In a previous communication [4] a method for obtaining nystagmograms with photographic amplification was briefly described. The technic is not perfect, but it probably represents a step forward in the study of optic nystagmus. It is sufficient to repeat here that Dodge's mirror recorder [5] is used; in this a concave mirror, pressed against the closed lid of one eye tangential to the underlying corneal surface, reflects a recording beam of light to moving photographic paper. The instrument records only the horizontal component of the eye movements. Rotated by the same motor that moves the photographic paper, a large wire mesh cylinder, concentric with the subject's head, carries letters and pictures of varying complexity. The cylinder, revolving around the subject's head, provides a moving visual field composed of stimuli of varying interest and complexity from the simple white lines of the wire mesh to a picture composed of eight faces. The visual field at any moment is limited to a sector comprising about 35 degrees of this surface, which is reflected in a framed mirror set at 90 degrees to the line of sight. This field differs materially from the relatively simple and monotonous stimuli provided by the alternate black and white stripes on the revolving drum used by most investigators and produces a characteristic normal nystagmogram. Since both field movement and photographic paper are synchronized, the record is not distorted by fortuitous changes in rate.

With the aid of this device it has been possible to supplement direct observations and to identify a considerable number of variations from

3. Cords, R.: Zur Theorie des optomotorischen Nystagmus. Eine Widerlegung Ohms, Klin. Monatsbl. f. Augenh. **77**:781 (Dec.) 1926; Ueber Hebelnystagmographie, Arch. f. Ophth. **118**:771, 1927.

4. Dodge, R., and Fox, J. C.: Optic Nystagmus: I. Technical Introduction, with Observations in a Case with Central Scotoma in the Right Eye and External Rectus Palsy in the Left Eye, Arch. Neurol. & Psychiat. **20**:812 (Oct.) 1928.

5. The particular form of mirror recorder utilized in this instrument was developed by a grant of the American Association for the Advancement of Science.

normal nystagmus. The gradual accumulation of data in carefully studied cases with verified neural lesions is being carried out by us with the dual hope that eventually additional light will be thrown on the neural organization of the eye movements, and also that our records will furnish more accurate data for tracing the course of certain neural lesions and their earlier diagnosis.

The records of normal subjects indicate the following characteristics of perfectly adaptive optic nystagmus for an angular velocity of the screen which is easy for the eyes to follow:

1. After an initial latency the slow or pursuit phases of the nystagmus begin abruptly with the pursuit fixation of some object of interest within the moving field.

2. The pursuit is commonly smooth and of an angular velocity closely approximating that of the moving object.

3. The amplitude of the slow phase or, in other words, the duration of pursuit fixation of any object is roughly proportional to the interest in the object and inversely proportional to the interest engendered by the peripherally seen objects, but other and at present unknown factors may modify the effect of interest.

4. The quick phase is normally a single saccadic movement of new fixation, of sharp onset and standard velocity. Each phase passes into the other without apparent latent period as though by some sort of anticipatory neural elaboration.

5. The amplitude of the quick movement may be said to depend on the distance between the actual object of pursuit and the object competing for new pursuit fixation. It seems to be a matter of attention with a strong bias of habit. Moreover, there is an underlying tendency for the eye to return to somewhere near its primary or central position.

6. Short saccadic movements of refixation, in both directions, occur within the pursuit movements and are more or less directly proportional in number to the complexity of the object of pursuit and the degree of visual analysis that it evokes.

7. There is usually a sharp onset of renewed pursuit at the conclusion of each refixation movement, again suggesting a pre-elaboration of pursuit innervation (figs. 1, *A*, *B* and *C*).

In considering these features of these two fundamental types of movement in optic nystagmus, it is important to keep in mind that only during pursuit eye movements is clear vision of a moving object possible. The quick or saccadic movements are momentary interruptions of clear vision,[6] of such short duration as to pass as unnoticed at the momentary blindness during a wink.

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6. Dodge, R.: Visual Perception During Eye Movement, Psychol. Rev. **7**:454 (Sept.) 1900; The Illusion of Clear Vision During Eye Movement, Psychol. Bull. **2**:193 (June) 1905.

Fig. 1.—All records were made by conjugate movements of a covered eye. They read from below upward. The actual direction of the eye movement is reversed, i. e., movement of the line of regard to the subject's right appears as an excursion to the left. Each figure represents about 8 seconds of a 32 seconds record. The speed of eye movement is shown by the obliquity of the excursions. Perfectly still fixation would be shown by a vertical line. Greater speed of the saccadic movements is also shown by relatively finer record lines. *A*, normal nystagmus, of subject E. H., a normal control; left eye recording. Slow pursuit phases of eye movement to the subject's left (responding to counter-clockwise movement of the objects) show as the heavier oblique excursions to the right. Rapid saccadic phases to the subject's right show as finer almost horizontal excursions to the left. The shorter relatively simple excursions *m-m'* indicate that the eye is following the mesh of the background. The longer more complex excursions indicate that the eye is following the more complicated pictures (eight faces, electric iron and parent with child, respectively). *B*, wink record of the subject, J. F., a normal control; left eye recording. Clockwise movement of objects. One wink is shown by a wide almost horizontal nasalward excursion with slower curved return to the original position, indicating a negative acceleration of eyelid recovery. *C*, lid tremors superimposed on normal nystagmus of R. D., a normal control subject; left eye recording. The lid tremors show as minute oscillation, waves of approximately equal velocity in both directions (especially clear at *t*), superimposed as overtones on the slow phases of a normal nystagmus. This is an exaggerated case, but few records are entirely free from eyelid tremors. *D*, rhythmic head movements, probably pulse of R. T., a normal control subject; left eye recording during still fixation. The pulse waves appear as waves of slightly larger amplitude—about one per second—superimposed on eyelid tremors. *E*, arrythmic head movement of H. L. in case 15. The record shows an arrythmic head tremor developing at *h. t.*, shortly after an initial period of inadequate pursuit. *F*, effects of fatigue of J. F., a normal control subject; right eye recording. Counterclockwise movement of objects. After eighteen revolutions of the screen at greater than optimum speed there appear moments when the pursuit becomes inadequate, and at one point the eye was still.

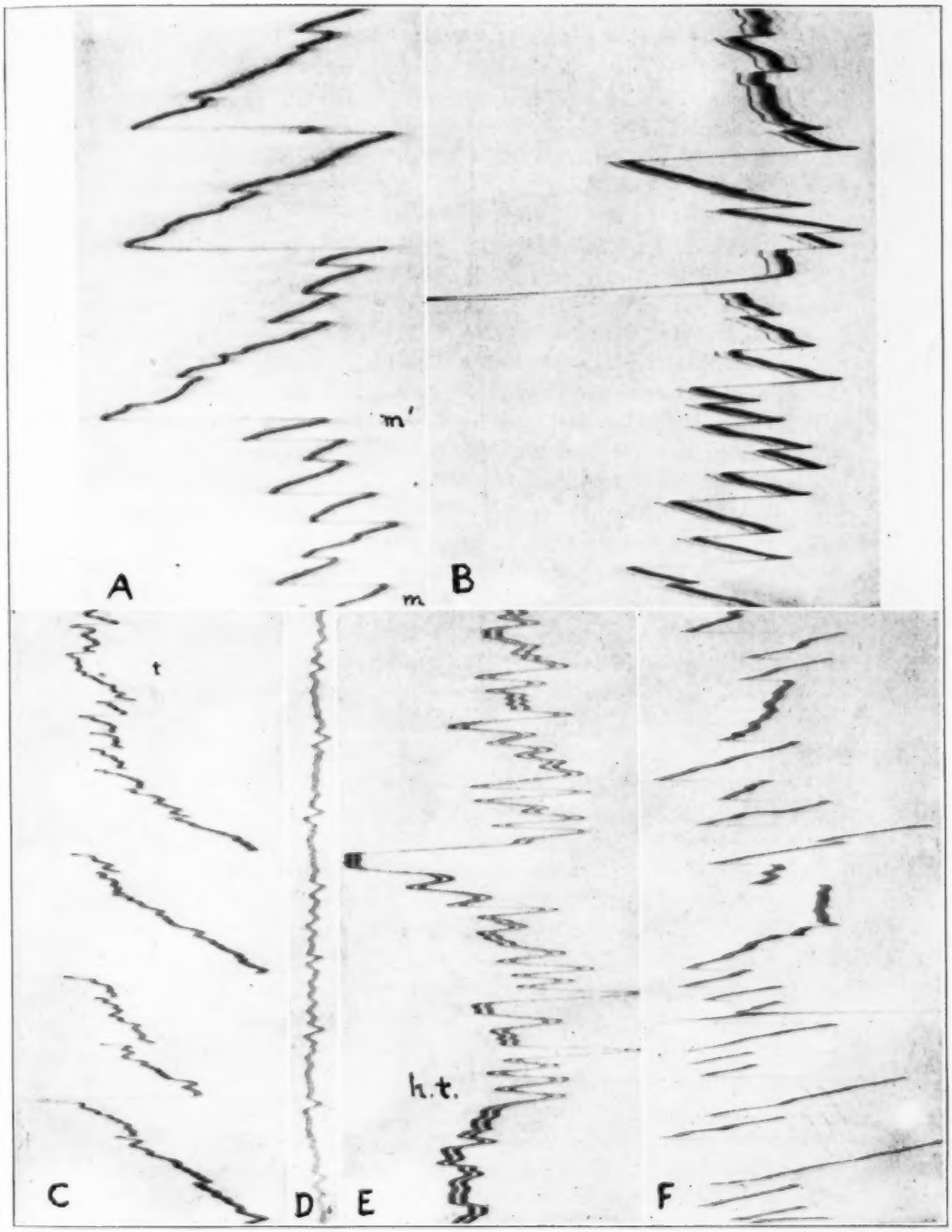

Figure 1

VARIANTS IN THE RECORDS ORIGINATING IN THE TECHNIC

The reader is referred to Dodge's original article[7] for a detailed description of the underlying principles of the mirror recorder for photographing eye movements when the eye is hidden under a closed lid. A mirror, which is pressed lightly against the lid over the center of the eccentric cornea, and which is entirely free to move, will always tend to assume a position tangential to the surface of the cornea on which it rests. For example, if the cornea moves clockwise underneath that part of the lid against which a mirror is resting, it will rotate the mirror counterclockwise. A recording beam of light from the mirror will consequently be displaced counterclockwise when the eye rotates clockwise.

Obviously, the photographic record obtained by such a system does not express itself solely in terms of the two fundamental types of adaptive eye movement concerned in optic nystagmus. All movements of the underlying lid, of the recording eye, or of the head, however slight, will be registered on the record. These sources of error might seem to be prohibitive and yet in actual practice such adventitious movements are usually readily identified in the record and interfere in no significant way with the interpretation of the nystagmograms.

Movements of the Eyelid.—Movements of the eyelid may produce arrythmic or rhythmic disturbances of the eye-movement record: 1. Wink records of the "lid reflex" type offer no difficulty of recognition to those who are familiar with their characteristics. They appear as rapid arrhythmic rotation of the mirror temporalward, due to drawing of the lid nasalward. They are of relatively large amplitude and return to the original position with negative acceleration (fig. 1, *B*). 2. Lid tremors are rhythmic disturbances of the record which occur both during still fixation and during eye movement (fig. 1, *C*). After considerable preliminary experimentation a useful eye-lid holder of adhesive tape has been employed which roughly has the shape of a figure "H." The uprights are attached to both the nasal and temporal parts of the upper lid and the skin over the malar prominences, thus keeping the lids snugly closed. This action is further helped by the narrow cross piece attached to the lower margin of the upper lid. The eye lashes are protected by a gauze strip attached to the lower half of the under surface of the adhesive tape. This device greatly reduces the lateral motility of the lid and notably diminishes disturbances of the record that originate in movements of the lids.

Fortuitous Eye Movements.—Fortuitous eye movements are of three types: eye drifts, fine oscillations or tremors and spontaneous nystagmus.

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7. Dodge, R.: A Mirror-Recorder for Photographing the Compensatory Movements of Closed Eyes, J. Exper. Psychol. **4**:165 (June) 1921.

1. All forms of eye-movement recorders, even under the most favorable technics, are less well adapted to indicate the primary central position or the exact deviation of the recording eye at any given moment than they are the direction and temporal duration of eye movement. Eye drifts are of slow velocity and of relatively long duration. They lend themselves easily to identification, appearing as gradual displacements of the total nystagmogram to the right or left side of the record. Similar gradual displacements are produced by head movement. Our present technic does not differentiate between them, but when the head is stabilized by a Helmholtz mouth bit, the head movements are relatively insignificant.

2. Fine oscillations of the eyes commonly occur and are readily recognizable as rhythmic waves in the control records of still fixation of the eyes which have been taken routinely in all our nystagmograms. They are indistinguishable from the lid tremors already described (fig. 1, *C*), but in either case do not interfere with satisfactory interpretation of the character of the adaptive eye movements. In fact, they frequently tend to disappear as soon as pursuit begins.

3. Spontaneous nystagmus appears in the records of still fixation, providing it occurs near the primary central position of the eye. In case spontaneous nystagmus does not develop in the resting central position, but only on lateral deviation, a control record may be obtained by eliciting both saccadic and pursuit movements of the eyes within the 30 degree zone of visual field permitted by the window in front of the seeing eye (fig. 3, *D*).

Head Movements.—Fixation of the head has always been one of the chief obstacles confronting those who have attempted to photograph the eye movements. The method employed in our work is that adopted by Dodge after long experience. The subject grips between his teeth a wooden spatula firmly fixed to the apparatus, a third point of support is furnished by a forehead rest. These measures have proved fairly satisfactory in eliminating gross head movements in all subjects who were sufficiently alert and intelligent to be reasonably cooperative.

Figure 1, *D* represents a rhythmic tremor synchronous with the pulse. This phenomenon is found rather frequently in the records of still fixation, and because of its periodicity, offers no difficulty of identification. "Pulse waves" tend to become less conspicuous during adaptive eye movements. They constitute one of the most regular of the conditions of faulty fixation.[8]

Figure 1, *E* shows an arrhythmic head tremor, developing shortly after an initial period of inadequate pursuit. One notes the irregularity of the head movements as regards amplitude and the returning and non-

8. Dodge, R.: An Experimental Study of Visual Fixation, Psychol. Rev., Monograph suppl. no. 4, plate 1, fig. (Nov.) 1907.

returning displacements of the record line. In general, the velocity of head movements may be said to be relatively slow, lying midway between that of the pursuit movements, as shown in our records, and the standard rapid velocity of saccadic movements.

Elimination of the errors due to movements of the head and eyelids is possible only by photographing the eyeball directly as in the method of the corneal reflection with a simultaneous head movement record. Even with an adequate head record, synergic movements of the eyeball and lid present difficulties in the exact analysis of the records.

ERRORS DUE TO MISPLACEMENT OF THE MIRROR

Success in obtaining records satisfactory for interpretation depends on the correct placing of the mirror recorder over the approximate apex or center of the cornea; otherwise the free movement of the mirror may be restricted in the direction of the displacement, and distortion of the eye-movement records appears. This consideration makes it difficult or impossible to apply this photographic technic to cases in which there is marked strabismus. In view of the fact that our pursuit nystagmograms presuppose conjugate movements in the nonseeing eye, it is advisable to exclude all cases of marked heterophoria, especially when the deviation of the closed recording eye would prevent the proper placement of the recording mirror. Consequently, in the preliminary clinical examination of each patient chosen for study, the behavior of each eye under cover is carefully noted.

NORMAL VARIATIONS OF OPTIC NYSTAGMUS

Numerous nystagmographic records have been obtained under uniform experimental conditions from normal subjects who did not show any clinical evidence of visual or ocular disorder or disease of the nervous system. Under these circumstances the nystagmographic picture conforms more or less closely to the pattern of what we have previously described as a "perfectly adaptive optic nystagmus." There are certain variations which appear either regularly in a series of records of one normal person or only occasionally in successive records of different subjects. Recognition of these variations is obviously important before an attempted interpretation of the nystagmograms of pathologic cases.

Inadequacy of Pursuit.—Even at a moderate speed of revolution of the cylindric screen during which the surface of the screen moves approximately 20 degrees per second at a distance of 50 cm., the pursuit phase may be more or less inadequate (top of fig. 1, *B*). This is evidenced by any variation in the slope of the pursuit curve, rarely by a brief still period at the beginning of the pursuit movement and more

often by a tendency to lag after pursuit has once begun. In the latter case, the pursuit phase assumes an S-wave nature with frequent corrective refixations of short duration. These refixations are not to be confused with the saccadic movements of larger amplitude which may occur in either direction during pursuit of an object of relatively greater complexity, and represent fixations on different points of the moving object of interest.

Inadequate pursuit of this type may manifest itself as a symmetrical disturbance to both directions of movement of the visual field. Our present data seem to indicate that this disturbance is more likely to occur while the seeing eye is pursuing temporalward.

Shortening of Pursuit.—Pursuit movements of the larger discrete objects regularly stand out in normal records by virtue of their greater amplitude as compared with the shorter pursuit movements of the mesh. Although this contrast is always maintained under normal conditions, there may be a general limitation of the extent of both mesh and object pursuit. This may express itself as either a symmetrical or an asymmetrical character, usually with a greater frequency during temporal pursuit of the seeing eye. The causes of the shorter pursuit movements elicited by the mesh are still obscure. We conjecture that it is partly a matter of peripheral visual acuity and partly a matter of decreased interest and attention.

Failure to Pursue.—The normal impulse to pursue is practically irresistible, provided the object of fixation moves. Occasional records reveal momentary periods of still fixation, which are likely to occur at the conclusion of a pursuit movement. The relative frequency of such episodes can be increased experimentally by the placement of a sufficiently bright, still object behind the semitransparent revolving screen. Some are probably conditioned by fixation of the frame which limits the visible area of the moving screen.

Saccadic Phase.—The velocity of the quick phase is remarkably constant for equal amplitudes in all normal nystagmograms, and the amplitude varies almost directly with that of the adjacent slow phase. Occasionally, the long return movements may be interrupted by a brief movement of pursuit, but in general both amplitude and frequency of the saccadic movements seem to be correlated with the amplitude and frequency of the pursuit phases.

INFLUENCE OF HIGH ANGULAR VELOCITIES AND OCULAR FATIGUE

The nystagmographic effect of high angular velocity of the moving visual field is under investigation in connection with the study of the thresholds of rotation. Increase in the velocity of the revolving screen

has a relatively slight effect on the amplitude of the pursuit movement but logically causes an increase in its angular velocity. Saccadic refixations within the object of pursuit tend to disappear with the higher velocities.

These features are readily distinguishable in figure 1, *F*, which is a record taken from the right eye of a normal subject after exposure to eighteen revolutions of the screen at the rate of approximately 40 degrees per second. The breakdown of pursuit under the influence of fatigue is clearly evident. In general, fatigue effects appear relatively later in normal subjects than might be expected. Except for the direct relation to the velocity of the moving visual field our observations as yet do not permit us to speak with definiteness regarding the characteristics of the phenomenon of fatigue.

VARIATIONS OF OPTIC NYSTAGMUS IN PATHOLOGIC CASES

A sufficient number of cases of disease of the nervous system have been studied to warrant the presentation at this time of some of the more important variations of the nystagmographic records which have been observed. The data here presented will not be discussed from the point of view of their possible bearing on differential diagnosis or on questions of neural integration and cerebral localization. Only the briefest reference to the underlying physiologic disorder will be attempted at this time.

The presentation of a complete case study in each instance is impracticable. Suffice it to say here that, as part of the complete neurologic examination, a thorough clinical study of the visual and ocular functions has been made in each patient. This examination included visual acuity, using a Snellen chart at 20 feet and Jaeger type at 18 inches (45.7 cm.), distance of screen; visual fields; ophthalmoscope examination; conjugate eye movements on command and pursuit of the moving finger; strabismus, and heterophoria (deviation under cover). The presence of a spontaneous nystagmus was carefully noted, whether positional in type and provoked only on conjugate deviation, or occurring in the resting central position. In addition, visual attention was studied by the method of moving two objects (forefingers) simultaneously before the patient in extreme lateral fields at equal distance from the fixation point.

The nystagmographic variations so far observed can best be classified primarily on the following basis: (1) as to whether they are symmetrical or asymmetrical, that is, whether they appear in response to both directions of movement of the visual field or to only one direction, and (2) as to whether the disturbance involves chiefly the pursuit (slow) or the saccadic (quick) phase.

CLASSIFICATION OF NYSTAGMOGRAPHIC VARIATION IN DISORDERS OF THE NERVOUS SYSTEM

I. Symmetrical nystagmographic variations—occurring during movement of the visual field in both directions.
   A. Disturbances of the pursuit movement.
      1. Shortening.
      2. Inadequacy of pursuit.
         (*a*) Slowing with corrective refixations.
         (*b*) Failure of mesh pursuit.
   B. Disturbances of the saccadic movement.
      1. Shortening.
      2. Dysmetria or "over-shooting" with correction.

II. Asymmetrical nystagmographic variations—occurring only or principally during movement of the visual field in one direction.
   A. Disturbances of the pursuit phase.
      1. Lag at onset and slowing with inadequate correction.
      2. Shortening.
      3. Absence of refixations within the object of pursuit.
   B. Disturbances of the saccadic phase.
      1. Failure to return to the central position.
      2. Interruption by abbreviated pursuits.

III. Asymmetrical nystagmographic disturbances provoked by conflict with a coexisting spontaneous nystagmus.
   A. Disturbances of the pursuit phase.
      1. Slowing with failure of correction.
      2. Elimination with periods of still fixation or reversal of direction.
   B. Disturbances of saccadic phase—reversal of direction.

IV. Erratic nystagmographic variations.

Symmetrical Disturbances of the Pursuit and Saccadic Movement (1A1, 1A2, 1B1).—Abnormally decreased amplitude of both pursuit and saccadic phases of the eye movements in response to both directions of movement of the visual field is shown in figure 2, *A* and *B*. These records represent the conjugate movements of the recording (right) eye, while the seeing (left) eye pursues objects on the revolving screen.

The case is that of a boy, aged 21, who was suffering from the characteristic complete clinical picture of myasthenia gravis. At the time of taking the records, four weeks after the onset of the condition, there was marked generalized weakness of all the ocular movements which was most striking for internal rotation and convergence. The lateral eye movements were not strictly conjugate, the inwardly moving eye in each case tending to lag behind its outwardly moving fellow. At the limit of the range of movement, estimated to be approximately 15 degrees temporalward and somewhat less nasalward, there developed an irregular positional nystagmus, followed by a drift of the eyes back to their central position. There was neither strabismus nor heterophoria. Notwithstanding the slight faults of conjugate movement as already described, the case was suitable for our nystagmographic technic.

Slow and inadequate pursuit and decreased amplitude of both pursuit and saccadic movements are obvious in each of the records. The movements per-

Fig. 2.—*A*, shortening and slowing of both phases of P. B. in case 9, myasthenia gravis with marked weakness of ocular movements, especially internal deviation; right eye recording. Clockwise movement of objects. The record begins with nystagmoid eye movements of small amplitude showing marked slowing of the saccadic phases. This is followed by a section in which the nystagmus is almost or completely lost, which in turn is followed by a section showing inadequate pursuit of the larger and more complex objects. *B*, shortening and slowing of both phases of P. B. in case 9 (same as figure 2, *A*); right eye recording. Counterclockwise movement of objects. The record shows inadequate and occasional disappearance of pursuit. *C*, inadequate slow pursuit of D. M. in case 17; multiple sclerosis with cerebellar disorder; right eye recording. Clockwise movement of object. This record shows the inadequate pursuit of four more complex objects with corrective saccadic movements in the direction of pursuit. *D*, dysmetria of saccadic movements, symmetrical, of J. H. in case 12; Friedreich's hereditary ataxia with marked cerebellar disorder; right eye recording. Clockwise movement of objects. The record begins with normal mesh pursuit nystagmus followed by pursuit of larger objects. All three long saccadic refixation movements overshoot the normal beginning of pursuit and return immediately, without intervening fixation, to the beginning of pursuit. This seems to us to be an unusually instructive picture of cerebellar dysmetria.

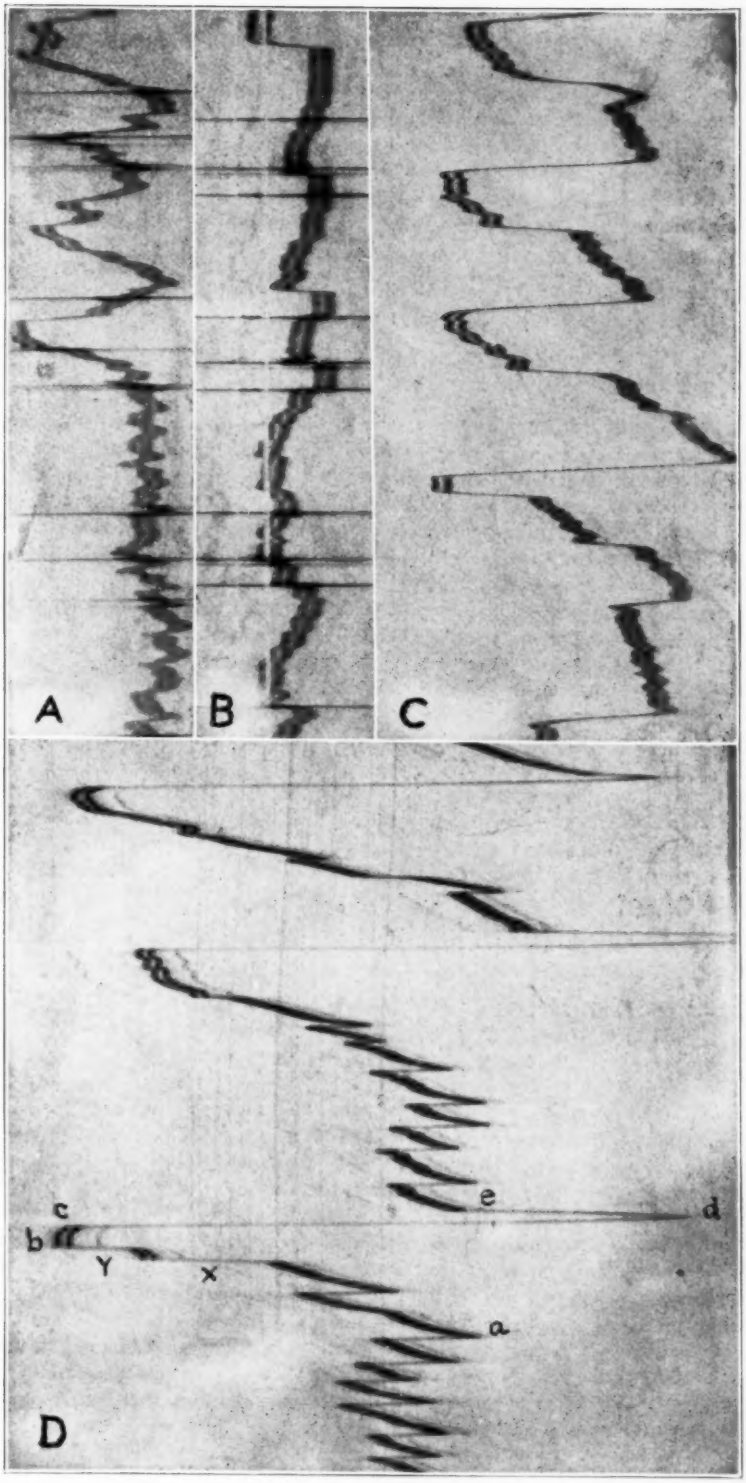

Figure 2

formed by the internal rectus of the recording eye show this more strikingly in each instance, i. e., saccadic in figure 2,*A*, pursuit in figure 2,*B*. The periods of still fixation at the end of brief pursuits in figure 2,*B* probably do not represent conjugate defects but rather the limit of excursion of the night internal rectus, while the seeing (left) eye is still pursuing temporalward by means of its stronger external rectus. The amplitude of the excursions still further decreased with development of fatigue.

Inadequacy of the Pursuit Movement—Symmetrical (IA2).—Figure 2, *C* represents the record obtained from the recording right eye of a male patient, aged 22, suffering from multiple sclerosis in a moderately advanced stage.

Neurologic examination indicated widely scattered damage to the central nervous system, producing a spastic ataxic paraplegia with marked impairment of proprioceptive sensation in the lower limbs. In addition, there was a bilateral cerebellar disorder, manifesting itself particularly in the performance of coordinated skilled acts and rapidly alternating movements of the upper limbs, more noticeable on the left side. Nystagmus was not noted with the eyes in their resting central position nor on lateral deviation within an angle of from 15 to 20 degrees on either side of the fixation point. In sectors in any direction beyond this, a coarse rhythmic nystagmus developed in which the quick component was always in the direction of deviation. The visual acuity was: right, 20/40, left, 20/30; Jaeger (18 inches) right, 3, left 2. There was slight temporal pallor of both disks, but no restriction of the visual fields.

The picture is that of an inadequate pursuit of the objects due to a lag of the eyes relative to the moving object. The lag is corrected by frequent refixations within the pursuit in the direction of objective movement. Pursuit of the mesh is entirely absent, apparently demanding a refinement of response not available. Since the response to counterclockwise rotation of the screen revealed an entirely comparable disturbance in the opposite direction, it has not been reproduced.

Defective mesh pursuit was also found in patients with impaired central vision as well as in those with a cerebellar disorder of fixation.

Dysmetria of the Saccadic Movement—Symmetrical (IB2).—An unusual record of dysmetria of the saccadic movement is shown in figure 2 *D*.

This record was obtained from the recording right eye of a male patient, aged 25, who was suffering from Friedreich's hereditary ataxia in a well advanced stage. In addition to the flaccid weakness, muscular atrophy and deformities in the lower limbs, there was a well marked cerebellar disorder involving all forms of coordinated motor activity. This was characterized by asthenia, retardation in contraction and relaxation, asynergia, dysmetria, gross intention tremor, deviation from the line of movement and inability to perform rapidly alternating movements. The eye movements both on monocular and binocular pursuit were of full range and conjugate in all directions. However, a severe disturbance of ocular stability was indicated by conjugate oscillations on attempted fixation, either central or on deviation in any direction. A photographic record of this conjugate oscillation was obtained from each eye during attempted still monocular fixation of the opposite seeing eye. It proved to be totally irregular, with eye movements of varying velocity and amplitude in both directions.

The most conspicuous abnormality in this record is the unusually large amplitude of the saccadic movement due to "overshooting." The details of one of these overshooting saccadic movements may be described as follows. The pursuit movement *a-b* was inadequate, showing two corrective refixation movements in the direction of pursuit, *x* and *y*. After a short terminal still fixation, the oncoming mesh initiates a saccadic refixation movement at *c*, which overshoots to *d*. The natural terminus was presumptively just below *e*. The reciprocal action of the antagonistic muscles, which should have stopped the eye, is delayed, causing an exaggerated saccadic movement which overshoots its natural terminus. The delayed action of the antagonists corrects the faulty overshooting by a rapid return to the preelaborated terminus at *e*, without latent period or intervening pursuit fixation. The preelaborated pursuit phase of this nystagmic oscillation begins from the point of the natural terminus after the overshoot has been corrected.

The record seems to us particularly interesting as it is probably a prototype of the faulty action of antagonistic muscles in the dysmetrias of cerebellar ataxia, and throws light on the innervation mechanism of the saccadic phase of nystagmus.

The "overshooting" in this instance is caused by the unimpeded contraction of the internal rectus of the recording right eye as a pseudo-conjugate movement to the actual saccadic movement of the external rectus of the seeing left eye. That this ocular type of dysmetria was a symmetrical type of defect was proved by the fact that a similar nystagmographic picture was obtained from the recording left eye in response to the opposite direction of rotation of the screen. Also, in general, all the nystagmographic records of this patient showed profound disorders of fixation concerned with both phases of response.

Asymmetrical Variations of the Pursuit Movement (IIA).—The term asymmetrical has been applied to those disturbances of the nystagmographic records which appear on movement of the visual field in one direction but not in the other. In addition to ocular palsies, they have been observed chiefly in cases of homonymous hemianopic visual defects—either blindness or inattention. The vital importance of the bearing of the whole question of hemianopia on optic nystagmus becomes immediately obvious to the worker in this field.

Case 21 is that of a woman, aged 27, who had a relative left homonymous hemianopia to within 10 degrees of the fixation point by perimetric examination. The visual acuity was: right 20/40, left 20/30; Jaeger, right 11, left 7. Examination of the fundi showed a bilateral papilledema of from 2 to 3 diopters with hemorrhages and exudate around the disk. There was slight weakness of the left external rectus on extreme deviation. The other clinical features of the case, including the postmortem observations six months after the nystagmographic records were taken, may be omitted here in view of our plans for detailed publication later.

Figure 3, *A* and *B* are records of the conjugate movements of the recording left eye and reveal a striking contrast in the response of the seeing right eye, depending on the direction of rotation of the screen. In Figure 3, *B* the objects

Fig. 3.—*A*, shortening of saccadic movements, asymmetrical of D. P. in case 21; left homonymous visual defect; left eye recording. Clockwise movement of objects. When the objects emerge from the defective side of the visual field there is disturbed pursuit and interrupted refixation movements. Compare figure 3, *B* (same case as in figure 3, *A*), counterclockwise instead of clockwise movement of objects. Nystagmus normal as objects emerge from the normal side of the visual field. *C*, interruption of saccadic movements by repursuits, asymmetrical, of J. C. in case 23; homonymous hemianopic inattention to the left; left eye recording; clockwise movement of objects. The pursuit of both the mesh and the larger objects does not show any noteworthy disturbance, but the saccadic excursions to the right show a series of short "groping" refixations instead of the normal simple sweeps. This was entirely consistent in all records and is unique in our experience. *D*, spontaneous nystagmus on deviation, more marked to left, in H. L. in case 15; multiple sclerosis; right eye recording; objects motionless. Spontaneous nystagmus appears in still fixation 15 degrees to the left of central position with quick phase to the left (right of the record). Still fixation 15 degrees to the right (left of the record) is not perfect, but does not show nystagmoid succession of slow and quick phases. *E*, conflict between spontaneous and optic nystagmus (same case as in figure 3, *D;* right eye recording counterclockwise movement of objects. The record shows the main trend of slow or pursuit excursions to the right (eye movement to left), but there are conspicuous exceptions. The picture is one of inadequacy, uncertainty and confusion, which is characteristic of rivalry for control of the final common path to the eyes. *F*, congruous spontaneous and optic nystagmus (same case as in figure 3, *D* and *E*). Clockwise instead of counterclockwise movement of the objects. The slow phases of spontaneous nystagmus are congruent with those of optic nystagmus. Under these circumstances the optic nystagmus appears almost normal in pursuit of the larger objects.

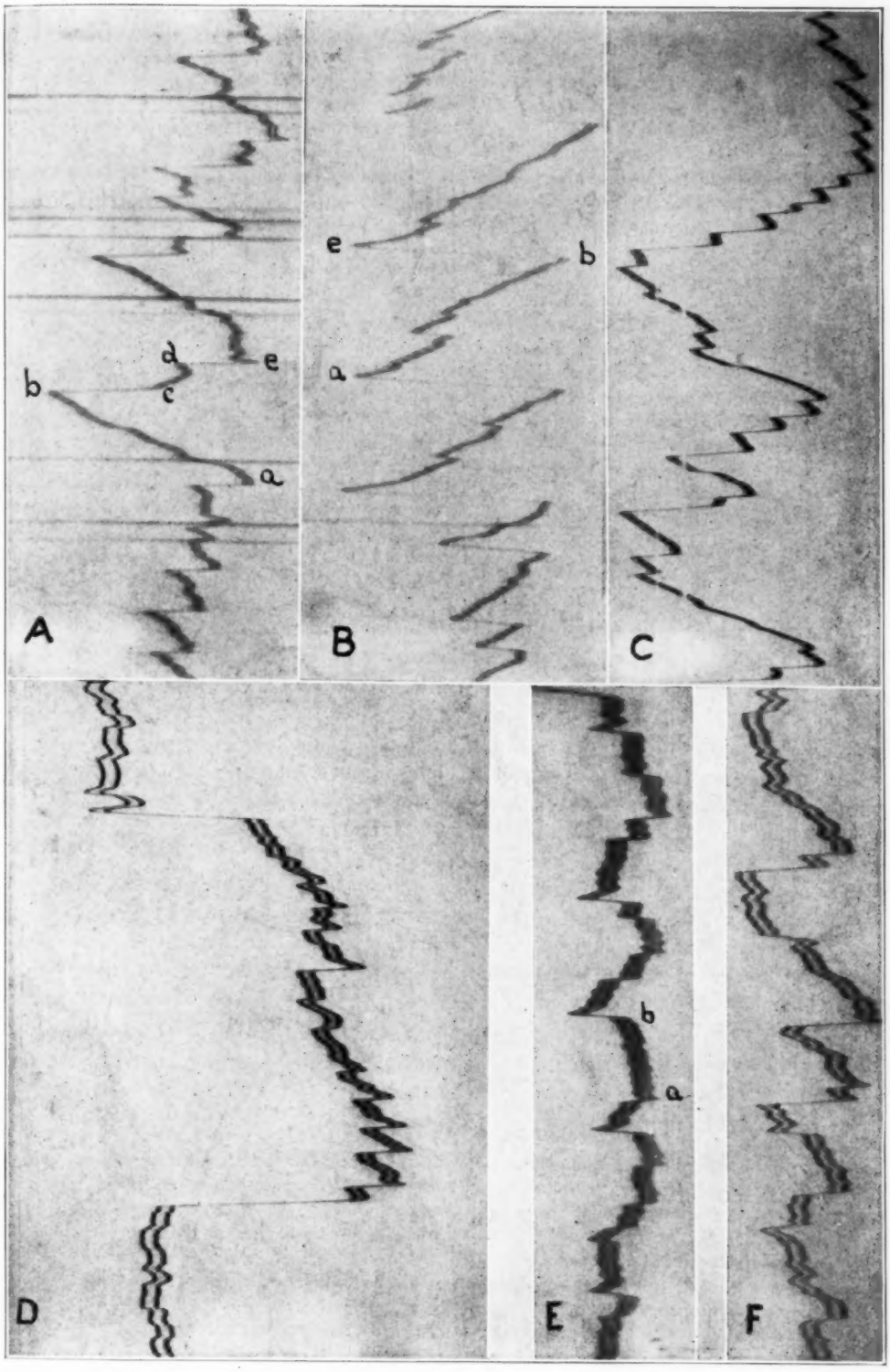

Figure 3

are emerging from the temporal or normal right field of the seeing right eye. A pursuit movement following one of the objects to the left starts at *a* and is broken by three short saccadic movements of refixation within the object of pursuit. At *b* another object of relatively greater interest, emerging from the right, stimulates an eccentric point on the functionally receptive nasal half of the retina, and evokes a saccadic movement *b-c* to the right, where a pursuit of this new object of interest starts at *c*.

If one compares this picture with that revealed in figure 3, *A*, a striking difference is apparent. The objects are now passing from the patient's left to her right, that is, emerging from the nasal or visually defective left field of the seeing right eye. The pursuit movements are, in general, defective as shown by a lag at onset and slow, inadequate pursuit without normal correction. There is also a defect or absence of refixations within the object of pursuit. These disturbances are undoubtedly directly related to the factors of inattention and difficulty of fixation of purely visual origin, as the objects of interest emerge from a region of practical blindness, preventing or delaying the preelaboration of the normal phases of the nystagmus.

Asymmetrical Variations of the Saccadic Phase (IIB1).—That disturbance of the saccadic movement in hemianopic visual defects likewise depends on the direction of movement of the visual field is also revealed in figures 3, *A* and *B*. Note the striking difference in the amplitude of the movements; the short saccadic movements in figure 3, *A* is, in part at least, a natural corollary of the shortening of each preceding pursuit phase. However, that this does not entirely explain the situation is shown by a study of that part of the record marked *a–c*.

In figure 3, *A*, *a* marks the beginning of a fairly long pursuit movement of an object passing from the patient's left to right. At the point *b*, when the seeing right eye is rotated externally a new object emerging from the left affords no stimulus or at least a relatively inadequate one to the functionally nonreceptive half of the retina. The shortening of the ensuing saccadic movement *b–c*, with almost still phase represented by *c-d* and final corrective saccadic movement *d-e*, all seem to represent a "groping" of the eye in an attempt to pick up the oncoming object, the real pursuit of which does not begin until *e*. There is also, undoubtedly, a tendency for the eye to return to its central position in the absence of an effective eccentric retinal stimulation.

Interruption of the Saccadic Phase by Interpolated Pursuits (IIB2).—Another type of asymmetrical disturbance of the saccadic movement is shown in figure 3, *C*.

The patient was a man, aged 57, with arteriosclerosis and hypertension, who had suffered with a rather profound disorder of visual attention to the left following a hemorrhage into the subarachnoid space six weeks previously. Although possessing normal visual acuity and not showing any defect of the visual fields, he consistently failed to appreciate the left of two simultaneously moving fingers, placed at equal distances to the right and left of the central point of fixation.

Figure 3, *C* represents the record obtained from the recording left eye when the screen is moving in a direction from the patient's left to right; that is, when the objects emerge from the side of impaired attention. The saccadic movement to the left is invariably interrupted by a succession of interpolated pursuits of brief duration, apparently serving the purpose of orientation in regard to the next object of interest competing for fixation and renewed pursuit. This picture seems to represent a prototype of the effect of disorientation in space peripheral to the point of regard.

Asymmetrical Disturbances Provoked by Conflict With a Coexisting Spontaneous Nystagmus (IIIA,B).—Figures 3, *D*, *E* and *F* show records taken from the recording right eye of a woman, aged 27, suffering from multiple sclerosis in a rather advanced stage, three years after onset of the disease.

Neurologic examination revealed signs of widespread damage to the nervous system including, in addition to the spastic ataxic paraplegia, a bilateral cerebellar disorder. This produced marked incoordination of the upper limbs for all types of motor activity and also a profound disorder in the attempt to maintain the sitting posture, characterized by a rocking of the trunk and tremor of the head (fig. 1, *E*). By means of adequate support this could be sufficiently inhibited to obtain satisfactory records of the eye movements.

Despite the evidence of optic atrophy on ophthalmoscopic examination, the patient's vision was only slightly impaired, Jaeger (18 inches), right and left 4. When the eyes were in their resting central position with distant fixation, they were practically still. Fixation at reading distance caused a rather irregular nystagmus the quick component of which was to the left. On left lateral deviation a vigorous rhythmic nystagmus developed, the quick component of which was to the left. On right lateral deviation a less active nystagmus developed, which corresponded to positional conditions, namely with quick component to the right.

Figure 3, *D* illustrates the spontaneous nystagmus clearly. Note the striking nystagmus following the saccadic movement of fixation 15 degrees to the left with gradual pull of the eye back to its primary central position. The absence of nystagmus in the record on fixation to the right is apparently due to the failure of development of nystagmus in this direction within the 15 degrees amplitude of movement permitted by the window of the screen in front of the seeing left eye.

Figure 3, *E* shows the resultant effect of a conflict between the spontaneous nystagmus and a superimposed optic nystagmus. Under this condition of counterclockwise rotation of the screen or movement of the visual field from the patient's right to left, the optically induced nystagmus should show a slow component to the left and quick phase to the right. Repeated records of this type of response, of which figure 3, *E* is a fair sample, showed a consistent failure to develop adequate pursuit even under the influence of repeated stimulation. There are a few feeble pursuit movements; during much of the time the eye is still, and in several places the slow movement, such as *a-b*, is in the opposite direction—to the right. Another striking feature is the complete absence of any correction of the defective pursuit. The saccadic movements may be in either direction, corresponding to either positional or optical conditions.

Figure 3, *F*, on the other hand, is the picture of an irregular but fairly effective optic nystagmus in response to counterclockwise direction of rotation. The pursuit movements, while varying in velocity and obviously inadequate in

much of their extent, are nevertheless almost consistently directed to the right. Also, with few exceptions, the quick movements represent true saccadic refixations to the left. Detailed description of the record is impracticable, but simple inspection shows a striking contrast with figure 3 *E*.

Both Dodge [9] and Carmichael [10] recently reported some interesting experiments in which they studied the effect of superimposing an optically elicited nystagmus on an induced vestibular nystagmus. When there was a conflict between the two as regards direction of the determining slow phase, regular movements of the eyes did not occur; when the direction of the slow phases coincided, the vestibular nystagmus was reinforced.

Erratic Nystagmographic Disorders (IV).—Records obtained from a patient suffering from a hypomanic form of excitement in the course of paresis illustrated an erratic type of behavior of the eyes, apparently determined by some profound disorder of the visual system. In our previous communication we pointed out the vital influence exerted by normal psychologic determinants on the mechanism of optic nystagmus. Erratic response of the eyes to a succession of moving visual stimuli must for the most part have their origin in pathologic disturbances of attention.

SUMMARY

Nystagmograms were taken by a mirror eye-movement recorder resting over the cornea on the lid of a closed eye. These records show conjugate deviations of the closed eye, as the other eye, in the axis of a cylindric screen, views more or less complicated, evenly moving objects.

Certain characteristic variations in the nystagmographic records of eye movement arose as a consequence of winks, tremors of the eyelids, pulse, head movements and fatigue.

Certain characteristic deviations from the normal picture of optic nystagmus occurred in the nystagmograms of the following disorders of the nervous system: myasthenia gravis with limitation of ocular movement; two cases showing a cerebellar disorder of fixation, one occurring in multiple sclerosis the other in Friedreich's hereditary ataxia; two cases with lesions in the right hemisphere causing left homonymous visual defects, in the first instance hemianopia, in the second instance inattention; multiple sclerosis with spontaneous nystagmus on lateral deviation.

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9. Dodge, R.: Adequacy of Reflex Compensatory Eye-Movements Including the Effects of Neural Rivalry and Competition, J. Exper. Psychol. **6**:169 (June) 1923.

10. Carmichael, E. A.: Optic Nystagmus, Proc. Roy. Soc. Med. **20**:53 (July) 1927.

A STUDY OF SPEECH DISORDERS IN FRIEDREICH'S ATAXIA*

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In classifying speech disorders, one differentiates three large groups: (1) those in which a lesion of the forebrain results chiefly in a disturbance of word comprehension; (2) those in which speech is affected in its motor component, and (3) those in which there is a defect of the purely mechanical, that is to say, muscular process of pronunciation although the comprehension and formation of the speech as an ingenious mental execution is absolutely normal. The nosologic classification under which these three groups of speech disorders are known includes sensory aphasia, motor aphasia and dysarthria. The names of Wernicke, Broca, Pierre Marie and Head are forever connected with knowledge of the pathology of speech. A large contemporary interest is directed toward the aphasias sensu strictiori, but comparatively little work has been done in the field of dysarthrias. Yet who as a neurologist has not noticed, in many a case of dysarthria, the obvious parallelism which exists between pronunciation and motor disturbances in other regions of the body? The speech disturbances in multiple sclerosis and in parkinsonism may serve as examples.

If one tries to realize a conception of pronunciative speech disturbances, one recognizes that the term dysarthria is not extensive enough to comprehend all the various disorders of this kind. If the internal speech is intact and no aphasic disorder is present, speaking does not mean merely a succession of letters, syllables and words, but an extremely complicated integration of well coordinated individual processes. These particular performances joined to a faultless and thoroughly individual character of the speech consist of: (1) the distinct pronunciation performed by the tongue, lips, palate and teeth ("euarthria"); (2) a clear phonation accomplished by the normal function of the larynx and pharynx, and (3) the intellectual and emotional part of the speech as expressed in accentuation, rhythm and varying pitch. To this aim, well integrated respiration and associated expressive movements of the face and even of the whole body are indispensable. The term dysarthria covers apparently only the disturbance of one functional part of this large unit. Thus it seems to

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me advisable to apply to the more extensive speech disorders the term dysphasia proposed first by Henry Meige in an almost identical sense.

Speech disorders in Friedreich's ataxia have already been described. Cassirer, for instance, in Oppenheim's Textbook of Neurology wrote: "The speech becomes slow, heavy, inaccurate, badly articulated and irregular." Schobb in Kraus-Brugsch's handbook said: "There is a characteristic bradylalia, sometimes connected with an explosive, scanning or monotonic inaccurate speech." Bonhoeffer, in his paper concerning the influence of the cerebellum on speech (1908), said, referring to a case of a cerebellar tumor: "The speech is slow, somewhat interrupted, accentuated in syllables, 'stumbling', and repeating; there are no bulbar symptoms, no ataxic or paretic disorders of the mouth, the tongue or the palate and the movements of the tongue are quick." He emphasized, however, a difficulty in changing the mouth from one position to another and designated this disturbance as an adiadokokinesis; he also found the speech to be slow and nasal. Both authors referred to destructions of the cerebellum by tumors or apoplexies.

Goldstein and Reichmann denied the assumption of a speech disorder resulting from adiadokokinesis alone but, stressing the ataxic components of this speech disorder, they pointed to the fact that decrease of speed, adiadokokinesis and ataxia may be present independently of each other in various body movements as well as in speech.

Gordon Holmes has contributed the best analysis of speech disturbances in cerebellar lesions. He wrote of unilateral cerebellar lesions as follows:

> The speech is usually slow, drawling and monotonous, but at the same time tends to be staccato and scanning. This gives an almost typical sing-song character and makes it indistinct and often difficult to understand. In many cases the utterance is remarkably irregular and jerky, and that of many syllables, especially, as Marie has pointed out, of those that end a sentence, tends to be explosive. Phonation is as a rule more affected than articulation, though both vowels and consonants are slurred and uttered unequally and irregularly. All classes of consonants too are affected, but articulation sometimes has a special nasal character and the labials particularly tend to be explosive. Another striking feature is the apparent effort necessary to utter a series of syllables or a sentence: the attempt is associated with excessive facial grimacing and speech has consequently a labored character that often recalls a pseudobulbar paresis. . . .

The observations of these investigators, especially the most complete study of Holmes, adequately describe speech disorders in cerebellar lesions. However, the study of four typical cases of Friedreich's ataxia together with that of a case of cerebellar ataxia on a degenerative arteriosclerotic basis convinced me of the pathognomonic importance of still another factor in this kind of a dysphasia, and suggested the attempt of a more thorough analysis of this speech disorder.

REPORT OF CASES

CASE 1.—*History.*—E, a man, aged 41, whose family history was unimportant and who was well until his twenty-fifth year, developed uncertainty in using his legs, especially in the dark. At the age of 28, the difficulties in walking increased and were associated with some pain in the legs, less in the arms, and clumsiness especially in the right hand. At 33, his speech became affected. He became "winded" in talking. For more than four years he had been confined to a wheel chair.

Examination.—The patient was unable to stand and showed a severe ataxia of the trunk when sitting. The cranial nerves were normal except for a bilateral horizontal nystagmus. He showed an abnormal respiration in that the respiratory movements were limited to the diaphragm and there was only very slight thoracic expansion. He had a marked thoracic kyphosis with a slight skoliosis to the right. All the tendon reflexes and the abdominal and cremasteric reflexes were absent; the Babinski sign was bilaterally positive. Tonus was normal in the arms but was decreased in the legs. There was a tremendous ataxia, more pronounced in the legs than in the arms, combined with a disturbance of deep sensibility; superficial sensation was intact. The ataxia interfered so much with active movements that other tests for cerebellar function were not applicable. The patient had typical "Friedreich feet."

CASE 2.—*History.*—P., a man, aged 30, was one of seventeen siblings, of whom one sister, now 28 years old, became diseased in the same manner as the patient at the age of 16. She had been an apparently normal child. The patient himself was, as he said, never absolutely normal; he was of poor physique, could not play like other children, and wrote with difficulty because of an uncertainty of his hands. The condition became worse at the age of 16, when he began to stumble over his feet. At 22, he could no longer walk nor use a cane or crutches because his arms were too weak and uncertain. Intellectually, he remained normal, but acquired some trouble with his sight. He had had a frequency of urination with urgency but no incontinence.

Examination.—The cranial nerves revealed a slight horizontal nystagmus to the left but no other pathologic signs. The patient was hardly able to sit up for any length of time; he soon began to sway and tended to fall backward. There was a moderate thoracic kyphosis. The mobility of the diaphragm was normal, but the expansion of the thorax measured in the mammary line was only 1 cm. Abdominal breathing was distinctly predominant. There was a marked ataxia in both arms and legs, but all active movements were possible. Tonus and gross power were almost normal. All tendon and abdominal reflexes were absent, but there was a positive Babinski sign on both sides. Of all sensory modalities, two-point discrimination and sense of position were disturbed. He showed a marked adiadokokinesis. The "Friedreich feet" were also present.

CASES 3 and 4.—*History.*—These two patients, St. and Th., were twin brothers, aged 18 years. In their eighth year (1918) both brothers, together with the other members of the family, were acutely ill with influenza. Up to that time they had been absolutely normal, but after they had been confined to bed for two weeks they could not walk or dress themselves. Their legs would not support them; their hands and head shook and were uncertain. Both patients gradually improved and were able to walk within two months and returned to school. In the subsequent years they became worse. Th. had had a serious breakdown, four and one-half years before examination, at which time he was bedridden for five months and a diagnosis of tuberculous meningitis (?) was made. Since then he has been able to

walk only a few steps with difficulty. St. was able to walk until eighteen months before examination, at which time he also became bedridden.

Examination.—St. was well developed; he could not stand and in an attempt to sit up showed a marked kyphoscoliosis of the thoracic spine and a pigeon chest. Respiration was performed almost alone by the diaphragm. There was a coarse horizontal nystagmus. The right pupil showed only a consensual reaction to light and there was optic atrophy on the right. In addition to motor disturbances of the face and tongue musculature to be described later, there were no pathologic symptoms of other cranial nerves. All the tendon reflexes were absent; there was a positive Babinski sign on both sides. The right arm was slightly spastic but tonus in the other limbs was not much altered. There was a very marked ataxia in the arms and legs but no loss of superficial sensation. He also showed typical "Friedreich feet."

Th. was unable to stand on his feet and had the same static ataxia as his brother. He had only a moderate kyphosis. The respiratory excursion of the chest was only 1 cm., but the movement of the diaphragm was normal. He had a marked horizontal nystagmus, but otherwise the cranial nerves were normal. The tendon and abdominal reflexes were absent. The Babinski sign was positive on both sides. All the movements were much impaired by the enormous ataxia in all limbs. The superficial sensation was normal in all phases. "Friedreich feet" were evident also in this patient.

The history of the last two patients, who, according to their appearance, are identical twins, is of special interest because of the onset of a so-called hereditary disease after a serious acute infectious disease. Even though the clinical picture leaves no doubt of the diagnosis it may be added that a younger brother, F., now aged 12, also became affected in a corresponding manner at the age of 10 years, but without having had an acute illness. Unlike the twins, he very gradually developed an awkwardness in the hands and feet. Now he shows a nystagmus and ataxia, but the reflexes are still present. A fourth brother, R., now aged 10, has only nystagmus without other symptoms. A girl, E., aged 8, and a boy, aged 17, show no symptoms at all. The mother and father are not related and as far as is known the family history is negative for any nervous diseases.

I may direct attention to some important facts: (1) The outbreak of "hereditary ataxia" in, at present, four of six siblings in a family without degenerative or other nervous diseases in the ancestry. (2) The identical age at which the ailment commenced in all four children. (3) The apparent rôle which an infectious disease, like true influenza, with an outspoken affinity for the central nervous system, plays in accelerating the course of an endogenous degenerative disease. Does it not seem as if special peculiarities of metabolic changes in a certain period of adolescence form the underlying basis on which physiologic claims of normal life lead to the slowly increasing dissolution of a part of the central nervous system, or on which a shock of the nervous system by an acute disease more or less destroys, at once, a system

already endangered? Maybe there is in diseases of this kind a critical age and there may exist a possibility of overcoming this dangerous point in development by a conscious avoidance of a great number of even physiologic demands. This should at least be tried in the younger children of a family in which a first case of Friedreich's ataxia has been diagnosed.

COMMENT

The speech disturbance in these four patients is practically the same, and shows only variations of a quantitative order. The description of speech disorders in cerebellar lesions as given by Holmes proves to be generally correct in my cases. The speech is "slow, drawling and monotonous but tends at the same time to be staccato and scanning." As to the other difficulties of articulation and phonation mentioned by Holmes, a more elaborate analysis explains a good many peculiarities of this disorder. A rather surprising fact is that the individualistic characteristics noticed in the normal speech of every person were found to be effaced to such a degree that one could hardly identify one of these patients by merely hearing him talk. In other words, the peculiarities of the dysphasia were much more outspoken than those of their individualistic pronunciation, phonation, speech melody, and the like.

Normally, the expression of the face reflects the psychic and intellectual processes involved in speech. The impairment of the mobility of the facial musculature injures the speech process as a whole, even if the volubility of the tongue is not markedly impaired. The monotony of the speech in cases of advanced parkinsonism illustrates this. When my patients are observed with regard to facial mobility, one sees that the integration of all the single movements of different parts of the face is disturbed. Thus the movements of the mouth are not normally transmitted to other muscle groups of the face and, on the other hand, active movements of one muscle group are not accompanied by a relaxation of muscles not intentionally involved in this act of motion. If either one of these patients tries, for instance, to sniff or to draw the angles of the mouth to the side, a much greater part of the face becomes involved than in a normal person.

Repeated grimacing also reveals another characteristic quality. There is a disturbance in the alternate motion rate of practically the same type as observed in the usual test for diadokokinesis. The time between two alternate motions is increased. One observes a similar disorder also in the tongue, but compared with the difficulty these patients have to move their lips quickly, the tongue seems not so much affected. One case shows especially an apparently normal volubility of the tongue. Though these patients do not show any difficulty in pronouncing different single vowels and consonants, the pronunciation of longer words, especially those with many lip sounds, is hindered by the adiadokokinesis

of the lips and to a certain degree also of the tongue. The lack of coordination of facial movements, together with an obvious adynamy of the facial musculature, is certainly responsible for some part of the speech disorder, at least the monotony and slowness are fostered by these difficulties in controlling facial expression. As Holmes said: "speech has consequently a laboured character."

Holmes said further: "phonation is as a rule more affected than articulation." This holds true in my patients, but the mechanism of the disturbance is not so simple. My patients when permitted to phonate uniformly showed a trembling of their voices which in its intensity was undoubtedly different from the slight tremor to be found frequently in normal voices.

The influence of the cerebellum on the vocal organs was subjected to an elaborate study by L. Bender. She concluded from her experiments in dogs that lesions of "those tracts which connect the cerebellum with the medullary nuclei, do produce motor disturbances of an ataxic type." She designated these disturbances as a form of adiadokokinesis of the vocal cords. A definite proof that in my cases there was such a disorder certainly depends on a registration of the vocal cord motion; this could not be done. The same holds true in regard to the movements of the soft palate, which are also of great importance in phonation. It does not seem to me beyond doubt that these disturbances can really be said to be a form of adiadokokinesis. Bender herself compared the accessory movements observed in the vocal cords of dogs with cerebellar lesions "with the action tremor which was noted also in the dog's neck and head . . . analogous to the so-called intention tremor." In my opinion, it is more likely that they express merely the pathologic change of the threshold of excitability in the vagus nuclei. The impairment of cerebellar function manifests itself in such a way that motor and tonic stimuli of a cortical, subcortical and medullary origin are not balanced by the peculiar function of the cerebellum. Thus the tremor results. The phonetic disorder, however, is not caused by the tremor of the vocal cords alone.

When these patients are observed, one notices that they breathe almost entirely with their diaphragms. Although, in an especially deep inspiration, they may use also the auxiliary muscles, under normal conditions the chest shows only a very slight expansion. The question arises whether this disorder is due to a primary distortion of the spine which is so common in cases of hereditary ataxia. Such a kyphoscoliosis, so outspoken as to influence the mechanism of respiration, however, is to be seen only in two of these patients; the other two show only a moderate kyphosis.

The usual explanation for the kyphoscoliosis in Friedreich's ataxia is a loss of tone of the chest and spinal musculature. One sees cases,

however, with kyphoscoliosis without marked hypotonicity. Thus one should think of the possibility that the impairment of function of the respiratory musculature of the chest, correlated with an incoordination of respiration—that is to say, an incoordination between the musculature of the chest and diaphragm—conditions this spinal disfiguration.

The second still more striking observation is that all four patients show another incoordination in respiration, one which occupies a higher level of coordinated movements and interferes with the normal integration of the motor act of speech itself. Pierre Marie has applied the term "dyspneumonie" to such disorders. This condition, briefly stated, consists of an inability to subordinate respiration to the meaning and inner sense of speech. Neither the strength of the voice nor the interruption of speech by respiration according to the meaning of the spoken sentences is regulated correctly. The patients usually start to talk with normal vocal intensity which decreases in a definitely steady fall to a state of exhaustion.

When the normal curves of speech and respiration are observed one sees how frequently persons must breathe in speaking longer sentences; they never destroy the sense of a well understood sentence. One can easily neglect disturbances of this kind which occur in a state of excitement. One sees also that a normal speech curve has a very marked up-and-down course, illustrating the senseful accentuation of words that have distinctly different meanings in their context.

Three curves from a larger number of normal speech curves illustrate this fact. Each upper curve in these tracings is the curve of speech obtained by means of a half closed system connected with a water manometer. The lower curves represent the abdominal respiratory movements taken with a tambour from a point between the xyphoid process and the umbilicus. The curves of respiration show that these three persons needed four, five and six inspirations for the text in question, but I have found normal persons who needed even up to ten. When the text is compared with its marks of respiratory interruptions one sees three examples of normal coordination of speech and respiration. This rather unscientific text was chosen, because it combines conditions which seemed to me important for this study. The patients liked to read it because it deals with a subject of universal interest; it contains some words not easy to pronounce and therefore likely to bring out a dysarthria, and finally its varying length of phrases and punctuation gives various possibilities of interrupting the flow of words by breathing, which would show either intelligible or senseless stops.

The three normal speech curves—Dr.G, H_2 and T_3—are shown in figure 1, and the corresponding respiratory interruptions, marked with G, H and T, above the text are to be seen below the tracing.

With these curves kept in mind, one sees in the three next curves (fig. 2) of a patient—E—with Friedreich's ataxia that he is apparently short-winded, needing from seven to ten inspirations for the test text. Such a fact, very common in Friedreich's ataxia, does not interfere with the integration of speech in a normal person as other curves have shown, nor is it essential for the disturbance in question since the second patient shows the same disorder without having a shortness of breath. This diminution of the capacity of the lungs finds its explanation in the marked kyphoscoliosis of this patient.

The irregularity of breathing seen primarily in the inequality of the intervals of the respiratory curve is significant for the pathology of his speech defect, and, much more important, there is an inequality which is not at all conditioned by the intelligent division of his speech, but which impresses one as being often absolutely senseless. Comparison of the curves in figure 2 with the text shown below illustrates this.

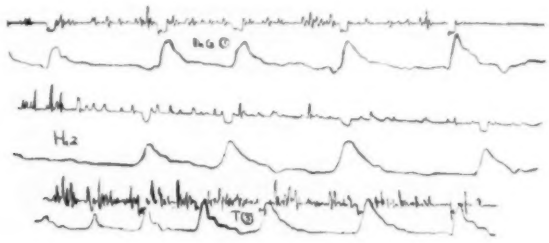

Fig. 1.—The radiator is in the smartest of Continental modes, its height accentuated by vertical shutters, its shapely shell brightly plated in chromium, richly embellished, and crowned by a symmetrical filler cap. The impressive length of the hood is emphasized by the brightly plated hinge from cap to cowl. Bodies are low set, gracefully paneled, fitted with short, military type metal visor of a distinctly new design.

The second unusual feature of his speech is the course of the speech curve; it does not show such a marked physiologically varying accentuation of words and syllables, but a preponderating tendency to start after each inspiration with some considerable force, ebbing down until the following inspiration. One could say that the respiration itself commands the flow of speech or that the speech loses its superiority over respiration.

In the curves of the second patient—P—(fig. 3), there are not any symptoms of shortness of breath, but nevertheless the same characteristics are present as in the first patient. Again, one observes the irregularity of breathing, not at all due to the sense of the text and changing in each test, contrary to the behavior of normal persons. The curve E4 with the abnormal inspiration after the first word of a new sentence illustrates this perfectly.

Figures 4 and 5 show six curves of the patients St. and Th. These brothers are, as the curves illustrate, rather short-winded. They need from nine to eleven inspirations. A survey of the texts (shown in figs. 4 and 5) shows the abnormalities of the respiration; in many instances the context of the sentence becomes severed by inspirations. Such an irregularity never would occur in a similarly short-winded normal person.

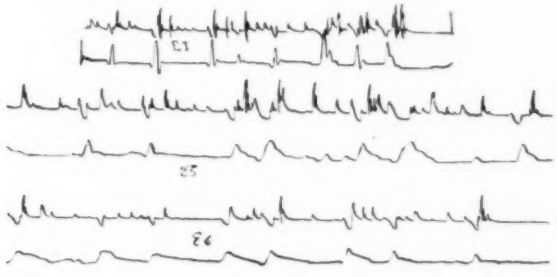

Fig. 2.—The radiator is in the smartest of Continental modes, its height accentuated by vertical shutters, its shapely shell brightly plated in chromium, richly embellished and crowned by a symmetrical filler cap. The impressive length of the hood is emphasized by the brightly plated hinge from cap to cowl. Bodies are low set, gracefully paneled, fitted with short, military type metal visor of a distinctly new design.

Experimental conditions mitigated against a successful record of these respiratory speech disorders in that I was forced to have the patients read the text several times before I traced the curves to be sure that the sense of the reading matter was really understood and to allow them to become accustomed to the apparatus. Such a procedure certainly means also a sort of training for the respiration and effaces a

good deal of the arbitrary interruptions of the speech by respirations such as were seen in the first attempts to read the text.

Another factor which must be obviated in a true reproduction of this speech disturbance is concerned with the use of a written text instead of the registration of free speech. Punctuation, as well as the beginning of a new sentence with capital letters, acts almost like a magnet and causes the reader to pause, less under the influence of intellectual reasons than under that of an optic stimulus. As a matter of fact, all my patients showed these symptoms much more clearly in their natural conversation, so that the diagnosis of a cerebellar lesion could be ascertained as soon as the speech had been heard.

If the various factors distorting the speech process in these patients are evaluated, then the faulty integration of respiration into the speech

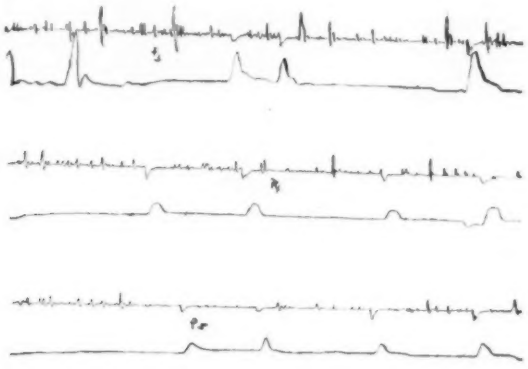

Fig. 3.—The radiator is in the smartest of Continental modes, its height accentuated by vertical shutters, its shapely shell brightly plated in chromium, richly embellished and crowned by a symmetrical filler cap. The impressive length of the hood is emphasized by the brightly plated hinge from cap to cowl. Bodies are low set, gracefully paneled, fitted with short military type metal visor of a distinctly new design.

act appears to me a very important one. The question arises as to what kind of a disturbance in a pathophysiologic sense is present. Is the disorder of respiration of the same category as those other disturbances of the speech process already mentioned? Furthermore,

since in these cases there is a defect of integration of a great number of single motor acts, normally combined to a faultless harmony, is one peculiar disturbance of motion the underlying cause of this disorder or do different factors act together to produce this pathologic picture?

REVIEW OF CEREBELLAR FUNCTIONS

To answer these difficult questions it seems advisable to review briefly the present knowledge of cerebellar functions. Kurt Goldstein has recently analyzed the existing theories of cerebellar physiology most completely and the following statements are largely drawn from his work, based on almost the entire clinical and experimental literature.

The cerebellum as a sensorimotor apparatus is intercalated in a highly complicated reflex mechanism. Since the largest part of its fiber connections are cerebellopetal—coming from telencephalic, diencephalic and mesencephalic centers—it seems probable that the cerebellum

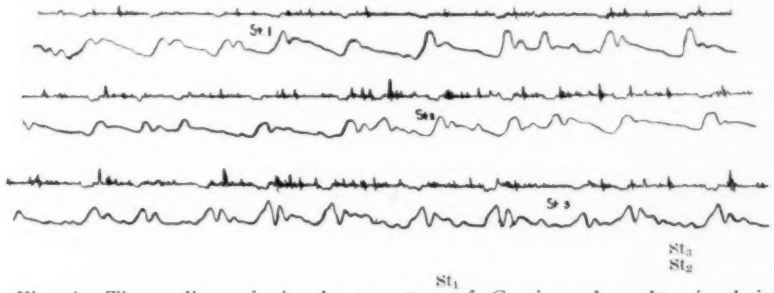

Fig. 4.—The radiator is in the smartest of Continental modes, its height accentuated by vertical shutters, its shapely shell brightly plated in chromium, richly embellished and crowned by a symmetrical filler cap. The impressive length of the hood is emphasized by the brightly plated hinge from cap to cowl. Bodies are low set, gracefully paneled, fitted with short, military type metal visor of a distinctly new design.

is subordinated to them. These anatomic data, together with clinical symptoms, suggest that the cerebellum apparently supports performances of other systems and has not an independent coordinative function of its own. Corresponding to the structural division of the cerebellum into paleocerebellar and neocerebellar portions, its functions are concerned

with the maintenance of the body equilibrium and symmetrical motions as well as with more asymmetric single movements.

Pathophysiologic studies of cerebellar lesions in man reveal the following symptoms: cerebellar ataxia, asynergy, hypermetry, retardation and adiadokokinesis. The ataxia results from more fundamental disorders, among which dysmetria or better, hypermetria, the rebound phenomenon, disturbance of antagonistic innervation and agonistic denervation, and the decrease of tone, particularly in voluntary movements, are of the greatest importance. These all reflect the pathognomic irregularity of innervation which together with its weakness and exhaustibility also results in tremor and abnormal fatigue in voluntary motion but never in the automatic movements.

As to the basis of adiadokokinesis, the most conspicuous manifestation of which is the prolongation of time between alternate movements, various underlying disturbances are important. These are

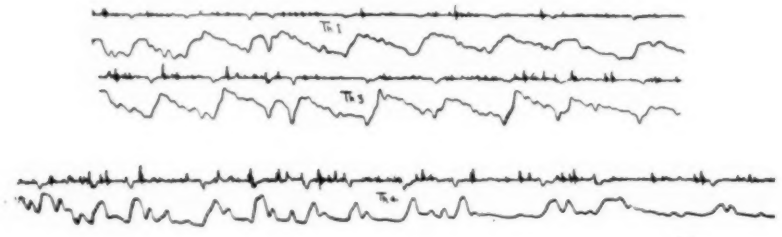

Fig. 5.—The radiator is in the smartest of Continental modes, its height accentuated by vertical shutters, its shapely shell brightly plated in chromium, richly embellished and crowned by a symmetrical filler cap. The impressive length of the hood is emphasized by the brightly plated hinge from cap to cowl. Bodies are low set, gracefully paneled, fitted with short military type metal visor of a distinctly new design.

retardation of innervation and denervation of the agonists and antagonists, the rebound phenomenon and impairment of successive induction. One should realize that the lack of cerebellar function as well as that of supracerebellar stimuli are combined in producing all these different symptoms.

Patients with cerebellar lesions show, besides this disturbance of single movements, also an impairment of the maintenance of direction of movements especially on the side of the lesion. Goldstein does not agree with Bárány who assumes that there are cerebellar centers which direct movements to the right, left, downward and upward, and even centers for parts of an extremity. Goldstein stresses the point that deviation of the limbs, particularly of the arm, always occurs outward and downward.

Sensory disturbances, such as the faulty valuation of weights and size, and the disturbed recognition of position are based apparently on a change in the threshold of excitability of motor, especially of lower motor, neurons, and thus on a deficient utilization of peripheral stimuli. In this connection, the observations of Hansen and Rich are of considerable value. By means of galvanometric measurement they found a prolongation of the refractory phase of the anterior horn apparatus on the side of cerebellar lesions in their patients.

In conclusion of the observations obtained in cerebellar lesions, Goldstein stated: All these symptoms represent deficits of functions (the vertigo and abnormal motions are only the subjective correlations of objective compulsory phenomena); true dynamic phenomena are caused by the liberation of certain subcerebellar apparatus from cerebellar influences. The deviation of the limbs and the backward falling are nothing but manifestations of the tendency to abduction and extension which has, as cases of total extirpation of the cerebellum have shown, its origin outside of the cerebellum, which itself responds to artificial stimulations with flexion and adduction. Those abduction and extension tendencies in cerebellar lesions are fundamentally comparable with extensor rigidity in decerebrate rigidity and other reflex automatisms in cases with impairment of voluntary motion.

Thus, these cerebellar symptoms represent the liberation of a primitive automatism by way of a lack of cerebellar counteraction, while normal cerebellar action tending to flexor and adductor movements is associated with nonautomatic, voluntary movements. Flexion and adduction are performed against the automatic abduction and extension and need much more the assistance of a co-innervation, the source of which apparently is the cerebellum. The degree of cerebellar cooperation in voluntary movements is regulated by proprioceptive stimuli. Flexion and adduction tendency on one side and extension and abduction tendency on the other is certainly not identical with innervation of flexor, adductor, extensor or abductor muscles but always deals with the aim of a movement.

The return of limbs into the most convenient position out of a compulsory inconvenient posture, as observed in cerebellar lesions, Goldstein also explained by a deficit of cerebellar activity which normally

co-innervates a cerebral action and arrests it automatically. Correspondingly, the deficit of cerebellar co-innervation influences the equilibrium of the body, the maintenance of which is to be regarded as a complicated function in which a large part of the central nervous system, especially the frontal brain, is involved. The disconnection of the vestibular apparatus from the cerebellum plays an important rôle in this disintegration of complicated functions. Goldstein denied the special coordinative function of the cerebellum for this purpose.

The explanation of the disturbances of single innervations encounters so many difficulties because of their insufficient reproductability in animals. Goldstein sees a fundamental factor for these disorders in the unbalanced excitability of the lower motor neurons toward voluntary impulses. Retardation of motor reaction, hypotonia, asthenia, uncertainty of movements, and tremor, all especially in volitional movements, are explainable in such a way. The difficulties are still greater if one attempts to elucidate the troubles in the regulation of the antagonists as enough is not known about its basis, namely, successive induction. Here also the loss of tone-increasing function of the cerebellum probably causes the disturbance of the highly intricated performances. Pathologic alterations of the influences on the cerebellum of an exterocerebellar origin lead naturally also to cerebellar disturbances which may, as in the case of an irritation of the cerebellum, lead to symptoms even like a hypertonia.

The abnormalities of the speech musculature of my patients as described appear to belong to the category of disturbances of so-called single movements. They are of a mixed symmetrical and asymmetrical type and manifest, therefore, a functional disorder of the paleocerebellum as well as of the neocerebellum. The symptoms observed in the facial musculature show a combination of ataxia, asynergia and adiadokokinesis, the cause of which, in accordance with Goldstein, is to be seen in the lack of the co-innervating influence of the cerebellum on the lower motor neurons, which also produces in my patients the adynamy of these movements and the trembling character of their phonation.

I assume that a severe lesion of the cerebellum leads, by means of an increase of the threshold of excitability of the lower motor neurons, to a prolongation of the time of motor reaction, to a delay in the start of a motor action and to a diminution of the motor strength. The disorder of antagonistic innervation is, according to Goldstein, in all probability also to be reduced to the loss of the tone-increasing influence of the cerebellum.

The respiratory disorder is obviously based on the same principles of a cerebellar disturbance, but reveals, on the other hand, somewhat more complicated mechanisms. The fact that intelligible periods of a

longer sentence themselves are not stimuli enough to provoke inspiration at determined points means very likely an increase of the threshold of the excitability also of these motor nuclei. Thus, these patients have to breathe either intentionally or because of want of oxygen, whereby the adynamy of the respiratory action may lead to an increase in inspirations. Volition and want of oxygen are apparently stronger stimuli than intelligible analysis of a sentence. How can one explain, however, the almost exclusive breathing with the diaphragm with the scant participation of the musculature of the chest? In endeavoring to explain this fact, I am conscious of the hypothetic character of this attempt.

A severe cerebellar lesion produces obviously also indirect effects by freeing other systems, as that of the cortex and subcortical ganglia, from cerebellar influences. The predomination of extension and abduction tendencies in the extremities in cases of cerebellar lesions is an example of this. It seems to me not improbable also that the positive Babinski sign in cases of cerebellar ataxia could be brought about in a similar way. The musculature of the chest as an organ of respiration is much more subject to volitional influences than the diaphragmatic respiration. It is partly what is termed an auxiliary musculature. One could assume that liberation from cerebellar co-innervation influences these normally coordinated respiratory systems differently, as spastic paresis may also do. The diaphragm moves in my cases by volitional stimuli or automatically, while, as described, psychogenetic factors easily fail to have this effect. The musculature of the chest responds, if at all, only to an abnormally strong impetus. Thus it seems to me that in cases of Friedreich's ataxia the cerebellar lesion brings about a separation of two physiologically coordinated but originally somewhat different motor processes; that it raises the threshold of excitability of the diaphragm and musculature of the chest but injures the chest respiration—as volitional to a great extent—still more by subjecting it to the released activities of other brain systems. I think one has to bear in mind also that though there is nothing like agonistic and antagonistic function in the diaphragmatic movement, the respiratory musculature of the chest can be regarded as composed of agonists and antagonists. Thus, disturbances of tone, excitability and muscular relaxation, all of which result in adiadokokinesis, asynergia and related phenomena, can be produced much more strikingly in the respiratory musculature of the chest, than in the diaphragm. To what extent a lack of proprioceptive reflexes from the diaphragm and still more the musculature of the chest causes the respiratory disorder cannot be decided. Be that as it may, I do not believe that the conception of simple ataxia of speech and respiration by loss of proprioceptive reflexes would further the knowl-

edge of this dysphasia more than an attempt of analysis as previously given.

I wish to emphasize that there is no reason to believe that in either case a mental disturbance conditions this speech defect. Moreover, all these patients astounded me because of their alertness and activity.

I think one should designate these speech disorders in hereditary ataxia, on account of their identity with those observed in cerebellar lesions of another origin, as "cerebellar dysphasia." Further studies, especially in cases of cerebellar lesions, will show to what extent the respiratory disorder of the speech is typical and may be dominating the other manifestations of cerebellar dysphasia. Investigation is also needed to determine whether there is a certain part of the cerebellum necessary to guarantee the normal coordination of all the muscle groups involved in speech, or whether—as seems to be more probable—every cerebellar lesion which impairs the function of the cerebellum as an entity does not injure also this highly complicated speech process.

THE HISTOGENESIS OF VON RECKLINGHAUSEN'S DISEASE *

S. H. GRAY, M.D.
ST. LOUIS

Verocay's [1] publication, in 1910, seemed to place the histogenesis of von Recklinghausen's disease on a new basis. He believed that the tumors arose from the cells of the sheath of Schwann. Four years later Herxheimer and Roth [2] thought that the connective tissue played a more important part in the origin of the disease than Verocay had ascribed to it, but it was not until recently that sufficient data had accumulated to make it possible to question seriously the interpretations of Verocay and his supporters. Mallory,[3] Penfield,[4] and Rhoades and van Wagenen [5] returned to the older conception, namely, that the tumors arise from connective tissue. On the other hand, Angola and Sabatini [6] and Hassin [7] published observations that support the conclusions of Verocay.

At Barnes Hospital, I had occasion to perform postmortem examinations in two typical cases of von Recklinghausen's disease, in both of which there had been sarcomatous changes. In one of these cases I studied several small tumors of the nerves in serial sections. The results that I obtained as to the origin of the tumors in the perineural connective tissue were so clear that I feel justified in briefly reporting the observations.

REPORT OF CASES

CASE 1.—H. W., a colored man, aged 37, had had multiple tumors for years (fig. 1). The terminal symptoms were cough, dyspnea and weakness; death was caused by a mediastinal tumor. The anatomic diagnosis was: multiple neurofibromas of the skin, intercostal nerves, sciatic plexus, stomach and duodenum; spindle cell sarcoma of the mediastinum, and passive congestion of all the organs.

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* From the Department of Pathology, Washington University School of Medicine, and Barnes Hospital.

1. Verocay, J.: Beitr. z. path. Anat. u. z. allg. Path. **48**:1, 1910.

2. Herxheimer, G., and Roth, W.: Beitr. z. path. Anat. u. z. allg. Path. **58**: 320, 1914.

3. Mallory, F. B.: J. M. Research **41**:349, 1920.

4. Penfield, W.: Surg. Gynec. Obst. **45**:178, 1927.

5. Rhoades, C. P., and van Wagenen, W. P.: Am. J. Path. **4**:146, 1928.

6. Angola, G., and Sabatini, F.: Ztschr. f. d. ges. Neurol. u. Psychiat. **103**: 496, 1926.

7. Hassin, G. B.: Neurinoma: A Case of Involvement of the Cauda Equina with the Clinical Picture of Bilateral Sciatica, Arch. Neurol. & Psychiat. **19**:1087 (June) 1928.

CASE 2.—J. H., a white man, aged 25, had had multiple tumors since childhood. The terminal symptoms were sore leg, gangrene of a toe and difficulty in voiding urine. Death was caused by a tumor in the left side of the pelvis. The anatomic diagnosis was: multiple neurofibromatosis of the skin, muscles and intestines, of the phrenic, sympathetic and intercostal nerves and of the celiac and sciatic plexuses; sarcomatous changes in the left sciatic tumor with erosion of the iliosacral joint and ileum; metastasis to the lung; metastasis in and rarefaction of the right pubic bone; gangrene of the left large toe; compression of the bladder; bilateral pyelonephritis and focal necrosis of the liver.

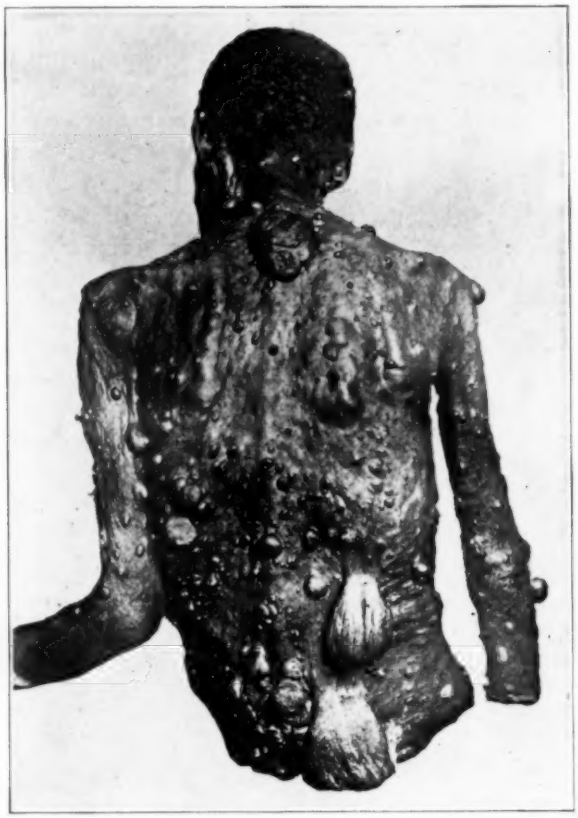

Fig. 1 (case 1).—A negro with von Recklinghausen's disease. The anatomic diagnosis in his case was multiple neurofibromas of the skin, intercostal nerves, sciatic plexus, stomach and duodenum; spindle cell sarcoma of the mediastinum, and passive congestion of all the organs.

Histologic Study.—Serial sections were made in case 2 through several of the small nodules attached to the phrenic and intercostal nerves. The sections included some of the uninvolved nerve contiguous to the small tumors. Figures 2, 3, 4 and 5 represent different sections through the tumor from the periphery toward the center. Figure 2 shows a beginning thickening of the perineurium, situated eccentrically about the nerve. Here the cells are spindle shaped with rather large,

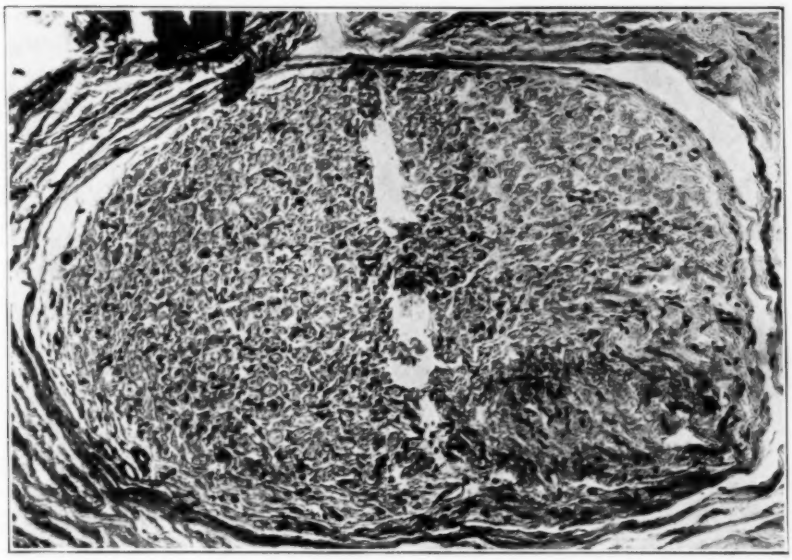

Fig. 2.—Edge of a tumor nodule attached to a nerve. The tumor appears as a thickening of the perineurium. Hematoxylin and eosin.

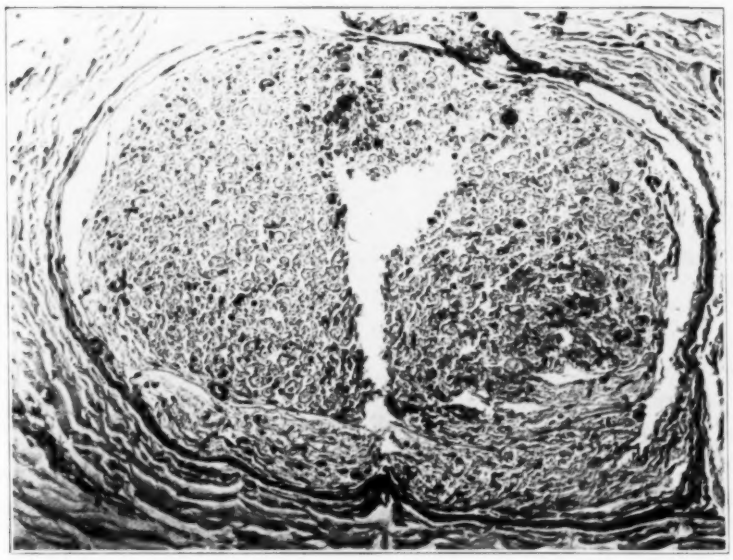

Fig. 3.—The cells of the tumor are shown arranged at right angles to the nerve. The separation of the tumor from the nerve may be noted. The tumor is eccentrically situated. Hematoxylin and eosin.

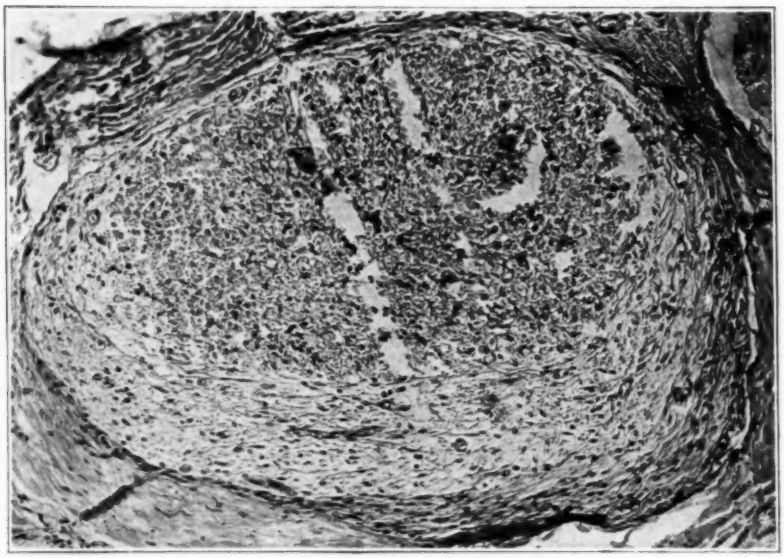

Fig. 4.—The tumor cells are shown densest at the periphery. The blood vessels are most numerous and largest at the perineurium. Hematoxylin and eosin.

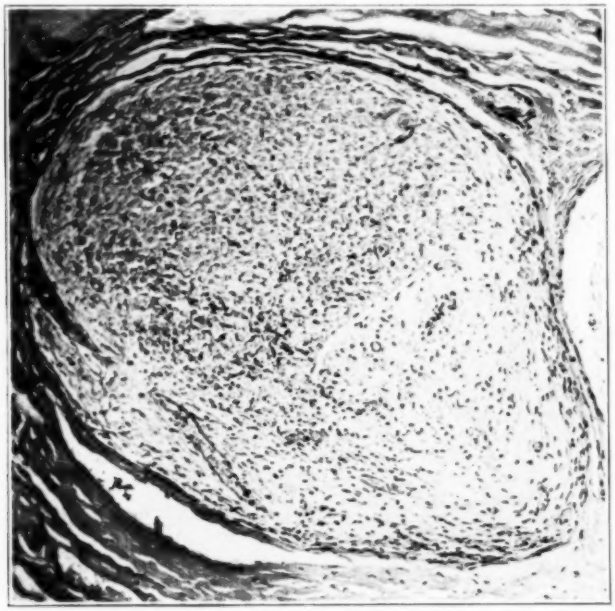

Fig. 5.—The widest part of the tumor. The cavity separating the tumor and the nerve may be noted, also the peripheral distribution of the blood vessels. Hematoxylin and eosin.

deeply staining elongated nuclei and definite fibrillar processes (fig. 6). The nuclei are fairly numerous. The long axis of most of the cells runs at right angles to the nerve. In figures 2, 3, 4 and 5, the tumor cells are seen as distinct from the nerve, and in figure 3 a narrow slit separates the nerve and the new growth attached to it. Blood vessels are noted in the tissue of the nodule in figures 4 and 5, and, especially in figure 5, the impression is given that these vessels have grown out from the capsule of the nerve. The same figures bring out another point of interest, namely, the greater density of the cells in certain areas of the periphery of the tumor.

Figures 7 and 8 picture two phases of another series of sections, in which the myelin is stained by the Weigert method. The edge of the nodule adjacent to the nerve, in places in which it is not involved in the tumor, shows practically no myelin; the tissue is here purely fibrillar. As one approaches the widest part

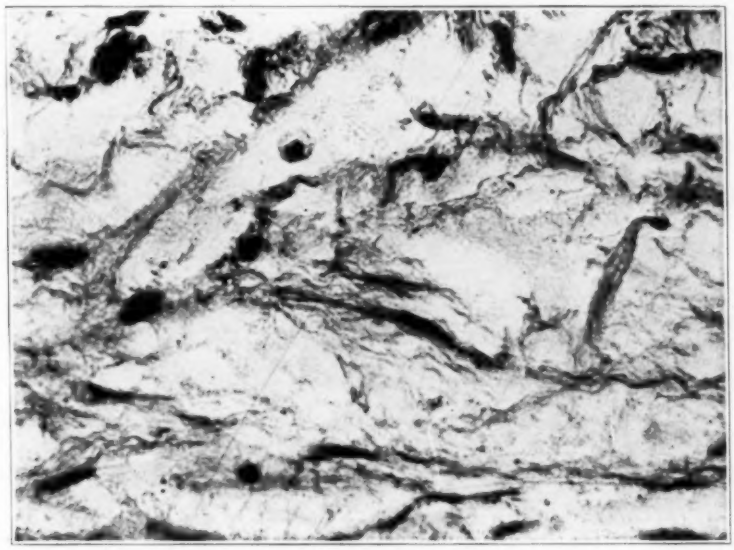

Fig. 6.—Cells of the tumor showing fibrillar processes. Mallory's aniline blue stain.

of the tumor, more and more nerve fibers are included in the mass, but not enough pressure is exerted to destroy the nerve, as may be seen from the fact that most of the myelin rings are intact. Also, where the tumor is thickest, it is surrounded by a band of connective tissue free from nerve elements.

The microscopic appearance of the tumor of the skin, of the muscle and of parts of the large masses in the mediastinum and pelvis that had undergone a sarcomatous change is shown in figure 9, which is a photomicrograph of a section of a subcutaneous nodule. The picture shows in different places a slight variation in the density of the cell and in the amount of fluid, some of the nodules appearing faintly green due to the large amount of fluid between the fibrils. Figure 10 represents a section through the sarcoma. The appearance is the same both in the primary sarcomatous tumors and in the metastases to the lung and bone.

Microscopically, the transformed tumor is a typical fibrosarcoma, as the photomicrograph shows.

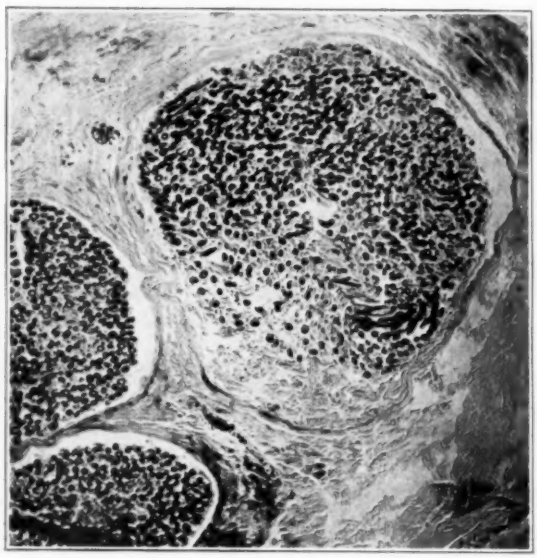

Fig. 7.—The edge of the tumor. Myelin is not found in the tumor nodule. Weigert stain.

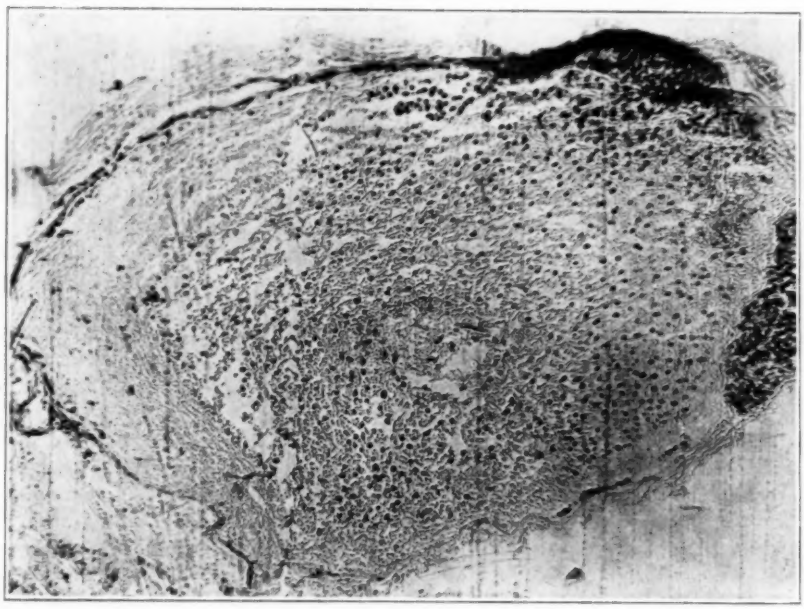

Fig. 8.—The middle of the tumor. Nerve fibers are shown included in the tumor; few of the nerve fibers are destroyed. The peripheral strand of tumor tissue does not contain myelin. Weigert stain.

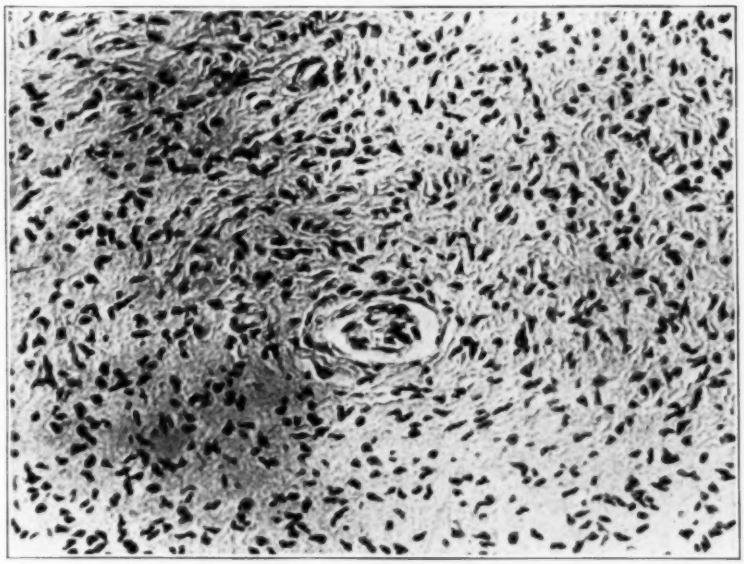

Fig. 9.—The microscopic appearance of the subcutaneous and muscular tumors. Parts of the sarcomas show a similar picture.

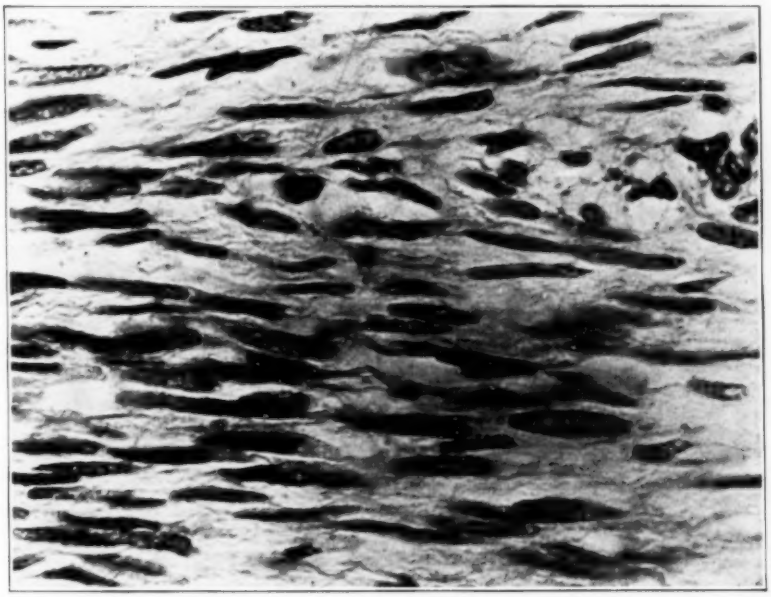

Fig. 10.—The sarcoma as seen microscopically in both its primary sites and metastases in case 2.

COMMENT

There can be no question but that, in these cases of von Recklinghausen's disease, a relationship between the cells of the sheath of Schwann and the cells comprising the tumors does not exist. The evident origin of the neoplastic tissue from the perineurium, the character of the fibrillar processes of the cells in the nonsarcomatous tumor and the nature of the cells in the sarcomatous tissue preclude such a conclusion. Although the embryologic origin of the perineurium is still unsettled, the character of the cells composing the tumors and especially the character and metastastic formation of the sarcomatous nodules make it appear more probable that this tissue is derived from the mesoderm.

Brooks and Lehman,[8] and Lehman[9] described changes of bone induced by a periosteal neurofibroma. In case 2, I had the opportunity of removing a piece of the right pubic bone; here I found rarefaction of the bone caused by a metastasis from the pelvic tumor.

SUMMARY

1. Two cases of von Recklinghausen's disease are presented, in both of which a fibrosarcomatous change had occurred.

2. In one of these cases, serial sections of one of the tumors show the tumor arising from the perineural connective tissue.

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8. Brooks, B., and Lehman, E. P.: Surg. Gynec. Obst. **38**:584, 1924.

9. Lehman, E. P.: Recklinghausen's Neurofibromatosis and Skeleton; Plea for Complete Study of the Disease, Arch. Dermat. & Syph. **14**:178 (Aug.) 1926.

PATELLAR TENDON REFLEX TIME IN PSYCHIATRIC AND IN NEUROLOGIC CASES *

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AND
JOHN M. DORSEY, M.D., M.S.
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Snyder [1] and Hoffmann,[2] each working with two normal subjects, found that the electromyographically determined patellar tendon reflex time ranged from 0.0080 to 0.0240 seconds. Travis and Hunter [3] studied this reflex time in eighty-seven normal adult subjects and found it to range from 0.0114 to 0.0268 second and to give a group average of 0.0197 second. Under normal conditions, they found reflex time relatively constant for the same person.[4] In another study of normal subjects we [5] found that alcohol markedly reduces patellar tendon reflex time. This last study offered further evidence toward the proposition that the greater and lesser irradiation of the higher centers of the central nervous system retards and facilitates, respectively, the activity of their subjacent levels. In a further attempt to strengthen or weaken the validity of this proposition, we undertook the present study of patellar tendon reflex time in psychiatric and neurologic cases.

METHOD

Essentially the same method as has been described in previous reports was used. The action currents were amplified by means of a three-stage, resistance-coupled amplifier and recorded by means of an oscillograph (Westinghouse, three-element portable). The amplifier was practically free from inherent disturbances. The supersensitive oscillograph element with which we recorded the electrical changes in the muscle was capable of responding within 0.00005 second. A General Radio low frequency oscillator furnished a time line of 1,000 cycles per second. The first electrode was placed over the motor point of the

* Submitted for publication, July 2, 1928.

1. Snyder, C. D.: The Latency of Knee-Jerk Response in Man as Measured by the Thread Galvanometer, Am. J. Physiol. **26**:474 (Oct.) 1910.

2. Hoffmann, P.: Beiträge zur Kenntnis der menschlichen Reflexe mit besonderer Berucksichtigung der elektrischen Erscheinungen, Arch. f. Physiol., 1910, p. 223.

3. Travis, L. E., and Hunter, T. A.: The Relation Between "Intelligence" and Reflex Conduction Rate, J. Exper. Psychol. **11**:342 (Oct.) 1928.

4. It varied rarely more than 0.0010 second.

5. Travis, L. E., and Dorsey, J. M.: Effect of Alcohol on Patellar Tendon Reflex Time, Arch. Neurol. & Psychiat. **21**:613 (March) 1929.

executant muscle. The second electrode was placed about ¼ inch (0.64 cm.) distal to the first. We designated as reflex time the time interval elapsing from the instant of stimulation of the tendon to the appearance of the electrical changes in the muscle.

REPORT OF OBSERVATIONS

CASE 1.—*Toxic-infective-exhaustive psychosis (catatonic syndrome).*

The patient was a white American woman, aged 19. At the time the first records were obtained the clinical picture consisted of catalepsy, flexibilitas cerea, parakinesis, muteness, stupor, subjective inaccessibility, no observable orientation and no discoverable insight. Enemas, catheterizations and tube-feedings were enlisted to assist vegetative functioning. Physical examination revealed undernourishment, pallor, a heart murmur, puerperal trauma and exaggerated deep reflexes. Laboratory studies revealed moderate secondary anemia and slight leukopenia.

The patient's mean reflex time as determined from a range of from 0.0050 to 0.0090 second was 0.0059 second.

Course.—On an antitoxic-infective-exhaustive regimen, there was a rapid uneventful recovery. At the end of four days she presented slight incertitude and irresolution, and was a little slow, but she was in relatively good rapport with the environment. She was friendly, cooperative, conversive and on an even emotional keel. Her habits of sleeping, eating and elimination were good. Slight exaggeration of the deep reflexes remained. Her mean reflex time as determined from a range of from 0.0175 to 0.0190 second was 0.0181 second.

After two months' residence in the hospital she had made a complete recovery both from a mental and from a physical standpoint and her mean reflex time as determined from a range of from 0.0180 to 0.0185 second became 0.0184 second.

Comment.—From this patient other significant data were obtained. During the stuporous state, a great increase in the frequency and in the amplitude of the oscillations of the action current occurred (fig. 1 *A*). The oscillations appeared at an individual rate of 520 per second in four or five groups of an approximate rate of 25 per second. Following recovery, these oscillations appeared at an individual rate of 300 per second in only a single group of relatively short duration (fig. 1 *C*).

CASE 2.—*Manic-depressive psychosis (manic phase).*

The patient was a white American woman, aged 47. At the time the first records were obtained she presented the basic manic symptoms of euphoria, hyperkinesis and ideational flight. The physical and laboratory observations were essentially normal. The mean reflex, as determined from a range of from 0.0200 to 0.0210 second, was 0.0206 second.

Course.—Four days later the case was considered as having improved to a hypomanic phase. The patient remained noticeably elated, talkative and overactive. The mean reflex time, as determined from a range of from 0.0185 to 0.0200 second, was 0.0191 second.

Fifteen days after the second series of records were obtained, the attending physician offered the diagnosis of cycloid personality to explain the clinical picture. At this time the patient had a mercurial disposition and presented an "up and down" attitude toward her environment. The mean reflex time, as determined from a range of from 0.0075 to 0.0190 second, was 0.0164 second.

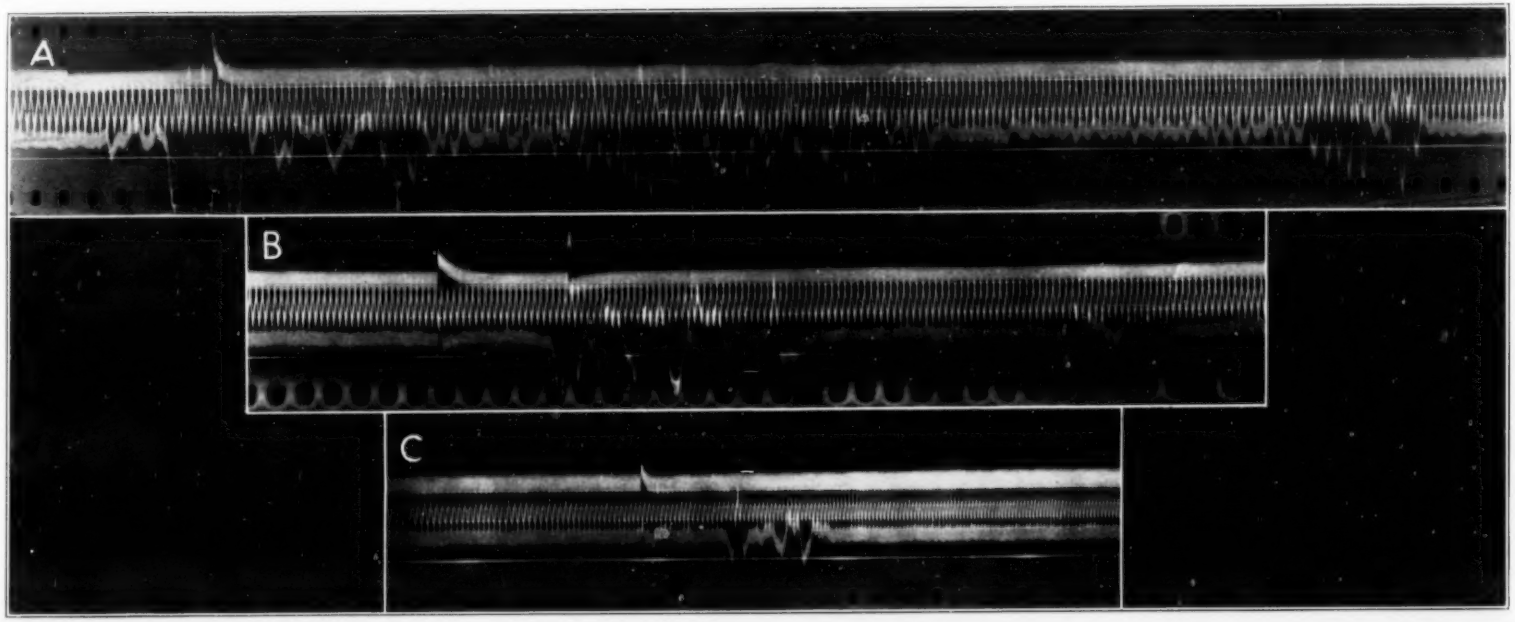

Fig. 1.—Records from case 1. Reading from above down in each record the first line is the signal line, the second the time line, and the third the action current line. *A* was taken during the stuporous stage. It shows an unusually short reflex time (0.0055 second), a relatively high frequency (520 per second), a relatively great amplitude of the oscillations of the action current, and the appearance of these oscillations in groups of an approximate rate of 25 per second. *B* was taken when the patient showed marked improvement (four days after *A* was obtained). The reflex time (0.0180 second) is greatly increased to approximate the mean reflex time (0.0197 second) of a large group of normal subjects. Although the amplitude of the oscillations remains increased, the frequency is greatly decreased (300 per second). No periodic groupings of the oscillations are found. *C* was taken after the patient had entirely recovered. It shows that the reflex time (0.0185 second) is further slightly increased and that the amplitude of the oscillations is greatly decreased. This frequency is approximately the same as that of the oscillations in *B*. They appear in a single group of relatively short duration.

CASE 3.—*Tumor of the left cerebrum.*

The patient was a white American man, aged 48. Just prior to the time the reflex time records were obtained, the patient presented a classic right hemiplegic syndrome, characterized by: exaggerated deep reflexes, absent patellar reflex (later a true Babinski reflex developed); absent abdominal and cremasteric reflexes; complete astereognosis; slight deep sensory loss over the shin and hand; choked disks; thick and almost unintelligible speech; spastic muscles; stiff tongue, and weak lower part of the face.[6] On the right (affected) side the mean reflex time, as determined from a range of from 0.0200 to 0.0210 second, was 0.0207 second. On the left side the mean reflex time, as determined from a range of from 0.0220 to 0.0240 second, was 0.0231 second. In addition to a shorter reflex time, the right leg gave action currents of much greater intensity (fig. 2, *A* and *B*).

Course.—Three weeks following operation,[7] the patient showed decided improvement. The confusion, ataxia, disturbed speech, spastic muscles and impairment of the sensorium had largely disappeared. On the right side the mean reflex time, as

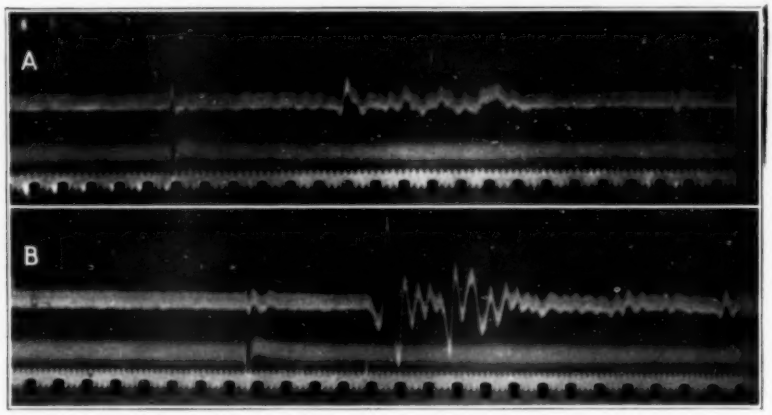

Fig. 2.—Records from case 3. Reading from above down in each record the first line is the action current line, the second the signal line and the third the time line. *A* is from the left leg, and *B* from the right (affected) leg. The latter shows a shorter reflex time and action currents of much greater intensity than does the former.

determined from a range of from 0.0220 to 0.0235 second, was 0.025 second. On the left side the mean reflex time, as determined from a range of from 0.0220 to 0.0235 second, was 0.0227 second. It is further significant that the two legs did not present differences in amplitude of the oscillations of the action current.

CASE 4.—*Spastic paraplegia (congenitalis)*

The patient was a white American man, aged 37, who presented the following clinical picture: flexion contracture of both knees and hips; talipes valgus; exaggerated deep reflexes; sustained quality of knee reflexes, and patellar clonus. On

6. Operation exposed a tumor limited, as far as determinable, to the left hemisphere.

7. The greatest portion of the tumor was removed.

the right side the mean reflex time, as determined from a range of from 0.0120 to 0.0170 second, was 0.0155 second. On the left side the mean reflex time, as determined from a range of from 0.0060 to 0.0165 second, was 0.0146 second. The records from both legs showed that the oscillations of the action current appeared in groups of an approximate rate of 20 per second. These groups persisted for as long as two or three seconds following the stimulation of the tendon.

COMMENT

This study offers a further contribution to the support of the proposition that the functional integration of the nervous system depends on the dominance of its more recently acquired levels over their respective substructures.

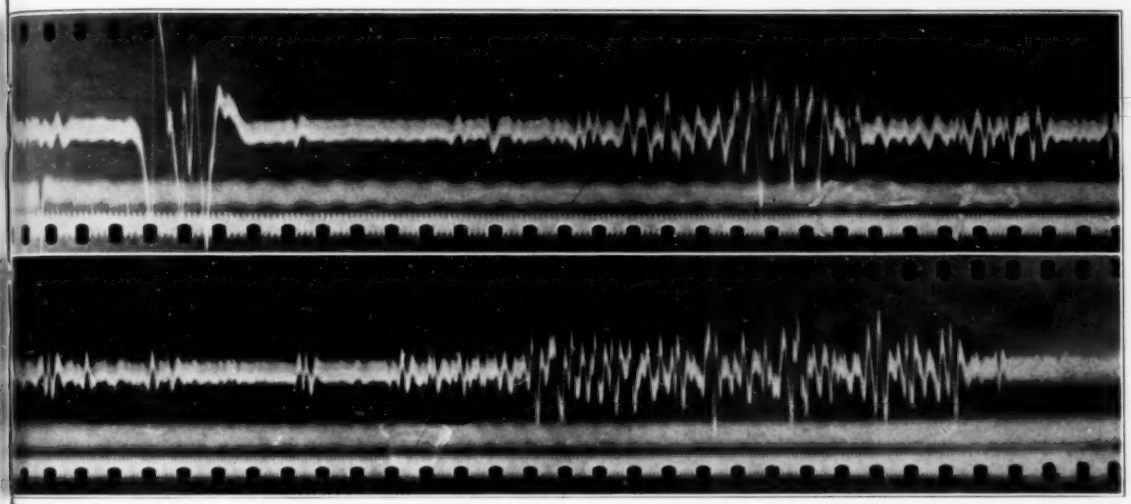

Fig. 3.—Record from the right leg in case 4. Reading from above down the first line is the action current line, the second the signal line, and the third the time line. It shows that the oscillations of the action current appear in groups of an approximate rate of 20 per second. The records of the left leg were similar to those of the right.

In an earlier study we found we could artificially affect the integration of this system by intoxicating it with alcohol. Because of the relative instability of the more recently acquired levels, a selective intoxication resulted. This afforded an opportunity for nice determination of the relationship between the levels of higher and lower order. In every case the relationship of dominance of the superstructure obtained. The intoxication throughout its progress expressed itself in the direction of depression. We interpreted this consistent effect to imply that alcohol, in its direct action on the central nervous system, is first and last a depressor. We found that the so-called period of excite-

ment resulted from the escape from domination and freedom of expression enjoyed, in turn, by each relatively less alcoholized neural level.[8]

When the diseased central nervous system was tapped by the same method that we employed in our work with alcohol, we obtained confirmation of our initial opinions. In case 1, in which stupor, flexibilitas cerea and muteness reflected arrested function of the higher levels, the peripheral arc enjoyed a striking freedom of expression. As the patient improved, the higher centers resumed their respective functions and the peripheral arc realized this in a return to a more subservient nature. For contrast to case 1 we selected in case 2 a patient presenting, as far as could be objectively determined, hyperfunction of the higher levels. We found that their apparent inordinate activity definitely depressed the peripheral arc. With improvement, manifested by a return to moderate exercise of the higher levels, the peripheral arc tended to resume its usual character. The wide range in the reflex time recorded at this period may be accounted for by exacerbations in the clinical picture. We selected case 3 to present a unilateral disease process of the higher levels because it provided a means for comparing in the same person the peripheral arcs of affected and unaffected sides. Again the impairment of the higher levels served to release the peripheral arc to less inhibited expression. When surgical intervention served to restore the higher levels to apparent health they resumed a natural relationship toward their dependencies. Case 4 presented a bilateral disease process of the higher levels that was reflected in an exceptional speed of the bilateral peripheral arcs.

CONCLUSION

We find that the common factor in all our cases is a constant relationship in the central nervous system between levels of the highest and lowest order of development. It expressed itself in increased subservience of the peripheral arc when the higher levels were inordinately active; in decreased subservience of the peripheral arc when the higher levels were inordinately inactive. Increased and decreased subservience respectively were denoted by the increase and decrease of the electromyographically determined patellar tendon reflex time. From this it would appear, in the central nervous system, that the transcortical neurons, or the highest levels of irradiation, are a functional part of the peripheral arcs, or the lowest levels of irradiation.

8. Unless the environment specifically occasioned them, the much described drunken euphoria and garrulousness were not evident.

THE MEASUREMENT OF CEREBRAL AND CEREBELLAR SURFACES

VII. THE MEASUREMENT OF VISIBLE AND TOTAL CEREBRAL SURFACES OF SOME VERTEBRATES AND OF MAN *

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CONTENTS

INTRODUCTION

In previous papers,[1] a definite method was established for the measurements of the cerebral and cerebellar surfaces. At the time, it was felt that a more thorough understanding of the cerebral surface of man could be accomplished by comparative studies in various vertebrates, from the lowest to man. This paper deals with the problem.

TECHNIC

In the study of these measurements, the same method of technic was followed as outlined in our previous paper. In the search for methods to eliminate possible sources for error, a new projection apparatus was devised at the suggestion of Dr. Arthur Weil, which gave an enlargement of the sections from 30 to 100 times.

Apparatus.—The apparatus consists of a modified Universal micrographic apparatus with a photographic mechanism removed (fig. 1). The apparatus consists

* Submitted for publication, Oct. 23, 1928.

* From the Neuropathology Laboratory, Montefiore Hospital, New York.

* This work was carried out under the auspices of the Hilda Stich Stroock Fellowship.

1. Kraus, W. M., and Ditto, M. W.: A Method of Measuring the Cerebral and Cerebellar Cortical Surfaces, Arch. Neurol. & Psychiat. **17**:193 (Feb.) 1927. Kraus, W. M.; Davison, C., and Weil, A.: The Measurement of Cerebral and Cerebellar Surfaces: III. Problems Encountered in Measuring the Cerebral Cortical Surface in Man, Arch. Neurol. & Psychiat. **19**:454 (March) 1928. Weil, A.: The Measurement of Cerebral and Cerebellar Surfaces: V. The Determination of the Shrinkage of the Surface of Different Vertebrate Brains, Arch. Neurol. & Psychiat. **20**:834 (Oct.) 1928.

of a Lilliput arc lamp with a clock fed mechanism operated with either a continuous or an alternating current. The clock work mechanism can be regulated to give a steady speed under all circumstances. Two carbons, one horizontal (positive) and one vertical (negative) form the luminous crater. The arc lamp is set in operation by turning a fiber wheel until the carbons come in contact. The second saddle stand carries an iris diaphragm provided with a holder for a cooling trough and a ground glass disk. The trough should invariably be filled with water and be entirely free from air bubbles. The third saddle stand carries the stage condenser. These condensers vary, depending on the lenses used. On the same saddle, there is a plate carrier for the slides. The fourth saddle carries a screw collar for the Summar lenses. The objectives used were 42, 80 and 100 mm. micro-Summar lenses. The slides are reflected on a projection screen.

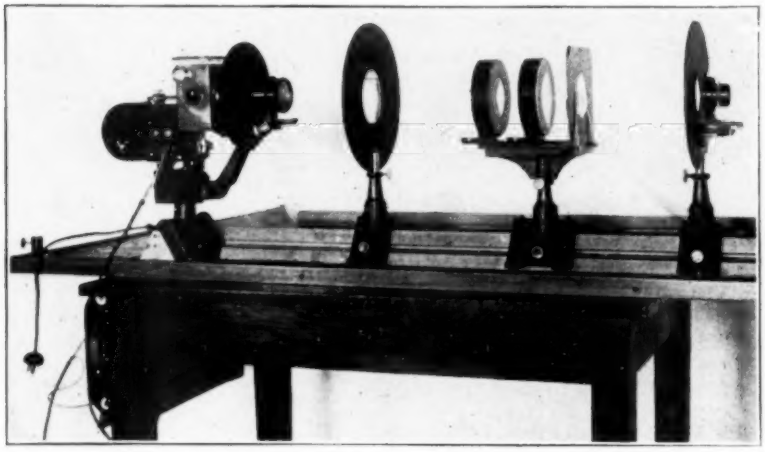

Fig. 1.—Universal micrographic apparatus with the photographic mechanism removed. A description is given in the text. On the extreme left is the Lilliput arc lamp. The second saddle stand contains the cooling trough. The third saddle carries the stage condensers and a plate which carries the slide to be projected. The fourth saddle carries the screw collar for the Summar lenses.

To obtain good results, it is necessary that all the optical components should be in perfect alinement. When projections are made with a 42 mm. micro-Summar lens, a stage condenser, with the inscription 64 mm. (plani-convex lens), is needed in addition to the large illuminating lens.

Material.—The animals used in the study of these measurements were: two opossums, two cats, two sheep, one fox, one dog, two monkeys and one child. When two brains of the same family were measured, they were cut in two different planes, i.e., horizontal and vertical. Sections cut sagittally were found inadvisable for use in measuring, for it was found difficult to cut the sections of both hemispheres at the same level.

The cutting, fixing and staining were done as outlined in our previous paper, with the exception that the sections were cut 50 microns thick and stained with both Weil's [2] and cresyl-violet methods.[2a]

Shrinkage.—The shrinkage of these brains throughout the process of fixing and embedding was taken into consideration. Table 1 and figures 2, 3, 4 and 5 show the losses in weight, volume and surface of the different animals.

On examining table 1, it is observed that the loss in weight, volume and surface of brains of animals belonging to the same family is about the same. The greatest loss is found in the brains of the opossum, the cat and the sheep. The least loss is in the brains of monkeys and

TABLE 1.—*Loss in Weight, Volume and Surface of the Brains of Various Animals Due to Shrinkage*

			Weight			Volume			Visible Surface (Surface Calculated as Surface of Globe)		
Animals	Position	Sex	Original Weight, Gm.	Loss in Weight, Gm.	Percentage Loss	Original Volume, Cc.	Loss in Volume, Cc.	Percentage Loss	Original Surface, Sq. Cm.	Loss in Surface, Sq. Cm.	Percentage Loss
Opossum	Horizontal	Male	11	7	63	11	6	54.5	8.72	2.32	36.3
Opossum	Vertical	Female	8	5	62.5	7	3.5	50	8.82	2.21	33.3
Cat	Horizontal	Female	30	15.5	51.7	23	14.5	56.5	26.12	6.51	33.4
Cat	Vertical	Female	26	12.5	48	27	14.5	46.2	35.14	9.61	37.67
Sheep	Horizontal	Male	112	55	49	110	52	47	79.38	19.8	32.5
Sheep	Vertical	Male	101	50.5	50	100	50.5	50	72.66	19.43	36.5
Fox	Horizontal	Female	38.5	15.5	40	39	15	38.4	30.23	6.17	25.6
Dog	Horizontal	Male	77	37	48	77	33	44.11	63.01	14.3	29.33
Monkey	Horizontal	Female	66.5	25.5	38.4	62	17	27.4	50.1	9.58	19
Monkey	Vertical	Male	66	27	39	61	19	38.3	58.08	8.45	19
Child	Horizontal	Female	445	205	46	440	185	42	243.6	58.61	31

man. In the brains of both stillborn and full-term children, there is a great deal of loss in the weight, volume and surface. When one considers that the fetal brain contains a large amount of water, the loss is expected at this period. The fox's brain should have shown a greater loss in the weight, volume and surface, but owing to the fact that the animal was extremely emaciated before death a great deal of dehydration took place in vivo.

CALCULATIONS

Opossum.—Two opossums were used. The brain of the male was cut horizontally and that of the female vertically. The horizontal sections were thirty-one in number and the vertical, thirty-three.

2. Weil, A.: A Rapid Method for Staining Myelin Sheaths, Arch. Neurol. & Psychiat. **20**:392 (Aug.) 1928.

2a. Every tenth section was measured. In order to know the total number of sections obtained, the number given should be multiplied by ten.

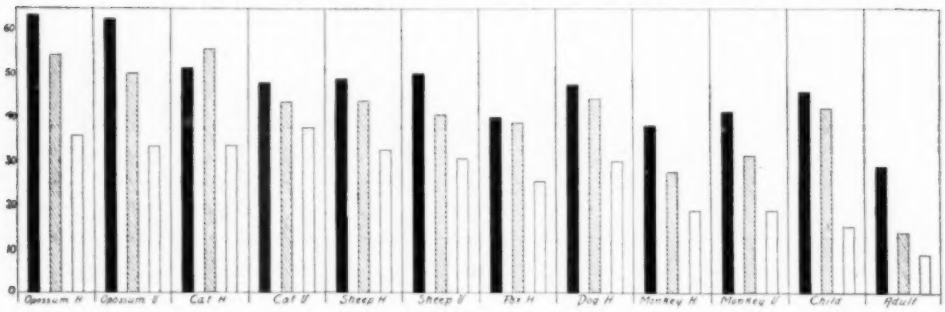

Fig. 2.—The final loss in weight, volume and surface of the brains of various animals during the processes of fixing and embedding. The unshaded columns indicate weight, the cross striped columns, volume and the blackened columns, surface.

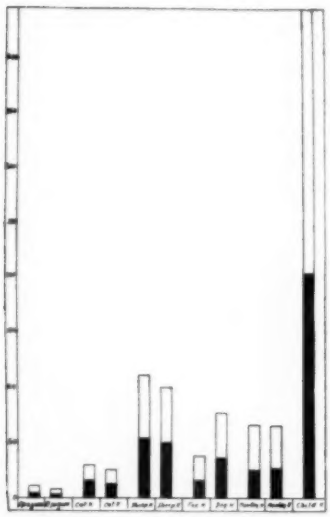

Figure 3

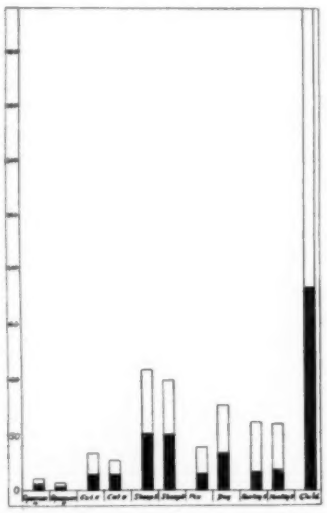

Figure 4

Fig. 3.—The weight of the brain prior to fixing and embedding and the loss in weight following shrinkage. The whole column indicates the original weight of the brain. The blackened part of the columns represents the final loss of weight of the brain and the unshaded portion of the column represents the final weight due to shrinkage.

Fig. 4.—The volume of the brain prior to fixing and embedding and the loss in volume following shrinkage. The whole column indicates the original volume of the brain. The blackened part of the columns represents the final loss of volume of the brain and the unshaded portion of the column represents the final volume due to shrinkage.

As observed from the table showing shrinkage (table 1) and figure 2, the two brains have lost 63 and 62.5 per cent in weight, 54.5 and 50 per cent in volume, and 36.3 and 33.3 per cent in surface due to shrinkage, respectively. As seen from these figures, the loss in volume approximates the loss in weight, while the loss in surface (36.3 and 33.3 per cent) is about one third of the original weight of the brain (figs. 3, 4 and 5).

Table 2 shows the visible, total and the averages of the perimeters of the right and left hemispheres, the ratio of the total and visible perimeters, and the visible surface areas.

TABLE 2.—*Perimeters and Ratios of the Visible and Total Surfaces of Opossum*

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
Horizontal—31 Sections													
123.40	104.89	3.98	3.38	7.36	58.94	51.24	1.90	1.65	3.55	2.092	2.045	2.068	6.40
Vertical—33 Sections													
87.82	86.62	2.66	2.62	5.28	51.70	50.40	1.56	1.53	3.09	1.70	1.70	1.70	6.61

TABLE 3.—*Visible and Total Surface Areas with and without Shrinkage in Opossum*

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
Horizontal													
3.42	2.98	6.40	6.94	6.31	13.25	36.30	4.66	4.08	8.72	9.50	8.56	18.06	2.07:1
Vertical													
3.33	3.28	6.61	5.64	5.56	11.20	33.3	4.44	4.38	8.82	7.51	7.42	14.93	1.68:1

Table 3 shows the visible and total surface areas with and without shrinkage, the percentage loss due to shrinkage and the ratios of the total and visible surfaces.

As is readily seen, the visible surface of these two cerebrums approximate each other, while the total surface of the brain of the opossum cut vertically is less. This is because the brain of the animal was younger and weighed less. As a result of this, the ratios of the total and visible surfaces of the brain cut vertically are less than those of the brain cut horizontally, as seen in table 3 and figures 3, 4 and 5. Figures 6 and 7 are a representation of the relationship of the visible and total perimeters.

The brains of two opossums were measured, the total surface being 18.06 sq. cm. in the horizontal series, giving a ratio of total to visible surface of 2.07:1; and 14.93 sq. cm. in the vertical series, giving a ratio of total to visible surface of 1.68:1.

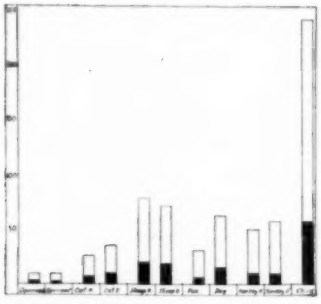

Fig. 5.—The surface of the brain prior to fixing and embedding and the loss in surface following shrinkage. The whole column indicates the original surface of the brain. The blackened part of the columns represents the final loss of surface of the brain and the unshaded portion of the column represents the final surface due to shrinkage.

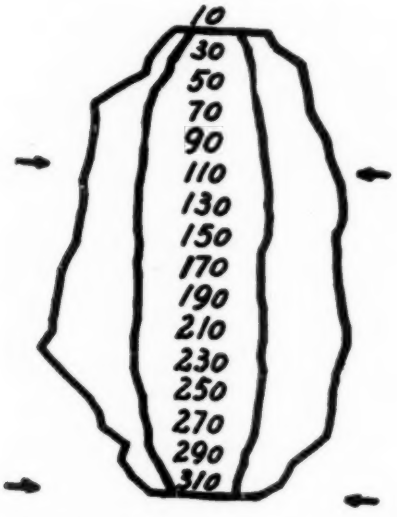

Fig. 6.—Horizontal section of brain of opossum. Graphic representation of the perimeters of the visible surface without the median longitudinal fissure and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.07 : 1. The hippocampus makes its first appearance at section 60 and continues to section 270. The cerebellum makes its appearance at section 100 and continues to the end. There are not many gaps in the different areas due to the small amount of fissuration.

Cat.—Two cats were used, both females; one brain was cut horizontally and one vertically. The horizontal sections were forty-three in number and the vertical, fifty-nine.

As observed from table 1 and figure 2, the two cerebrums have lost 51.7 and 48 per cent in weight; 56.5 and 46.2 per cent in volume, and 33.4 and 37.67 per cent in surface, respectively. As seen from these figures the loss in volume approximates the loss in weight, while the loss in surface (33.4 and 37.67 per cent) is one third of the original weight of the brain (figs. 3, 4 and 5).

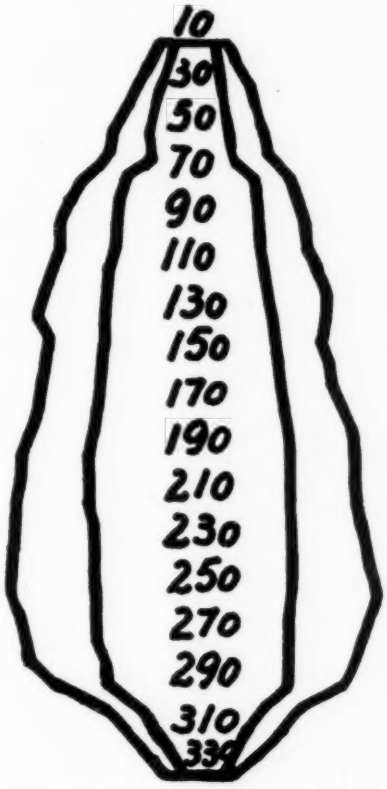

Fig. 7.—Vertical section of brain of opossum. Graphic representation of the perimeters of the visible surface, without the median longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 1.68 : 1. The hippocampus makes its first appearance at 100 and continues to section 290. The cerebellum in this series does not make its appearance due to the fact that the brain was cut vertically.

Table 4 shows the visible, total and the averages of the perimeters of the right and left hemisphere, the ratios of the total and visible perimeters and the visible surface areas.

Table 5 shows the visible and total surface areas with and without shrinkage, the percentage loss in surface due to shrinkage and the ratios of the total to the visible surfaces.

The visible and total surfaces of the brain cut horizontally are less than those of the one cut vertically. The ratios of the visible and total surface areas are approximately equal in both brains. Figures 8 and 9 are a representation of the relationship of the visible and total perimeters of the two brains.

The brains of two cats were measured. The total surfaces were 58.76 sq. cm. in the horizontal series, giving a ratio of the total to visible surface of 2.25 : 1, and 75.25 sq. cm. in the vertical series, giving a ratio of 2.14 : 1.

Sheep.—Two sheep were used, both males; the brain of one was cut horizontally and the other vertically; the horizontal sections were fifty-two in number and the vertical sections, thirty-three.

TABLE 4.—*Perimeters and Ratios of the Visible and Total Surfaces of Cat*

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
Horizontal—43 Sections													
353.28	371.49	8.23	8.64	16.87	160.91	163.67	3.74	3.80	7.54	2.20	2.27	2.23	19.61
Vertical—50 Sections													
390.28	424.23	6.61	7.19	13.80	197.83	183.78	3.34	3.12	6.46	1.98	2.30	2.14	25.53

TABLE 5.—*Visible and Total Surface Areas with and without Shrinkage in Cat*

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
Horizontal													
9.74	9.85	19.61	21.39	22.44	43.83	33.4	12.99	13.13	26.12	28.67	30.09	58.76	2.25:1
Vertical													
13.40	12.13	25.53	26.35	28.28	54.63	37.67	18.90	16.72	35.14	36.02	38.96	75.23	2.14:1

As observed from table 1 and figure 2, the two cerebrums have lost 49 and 50 per cent in weight, 47 and 50 per cent in volume and 32.5 and 36.5 per cent in surface, respectively. As seen from these figures the loss in volume approximates the loss in weight, and the loss in surface (32.5 and 36.5 per cent) is a little more than one third of the original weight of the brain (figs. 3, 4 and 5).

Table 6 shows the visible total and the averages of the perimeters of the right and left hemispheres, the ratios of the total and visible perimeters and the visible surface areas.

Table 7 shows the visible and total surface areas with and without shrinkage, the percentage loss due to shrinkage and the ratios of the total and visible surfaces.

The visible and total surface of the brain of the sheep cut horizontally is slightly greater than that of the one cut vertically. The ratios of the total to the visible surfaces are approximately equal. Figures 10 and 11 are a representation of the relationship of the visible and total perimeters.

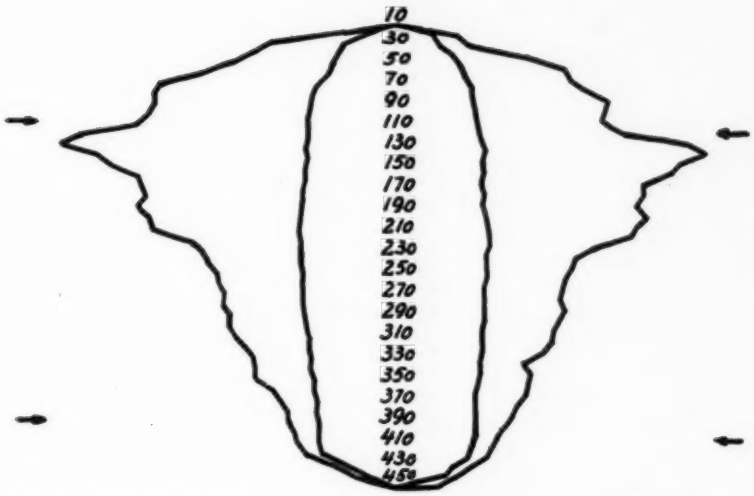

Fig. 8.—Horizontal section of brain of cat. Graphic representation of the perimeters of the visible surface, without the medium longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.25 : 1. The cerebellum makes its appearance at 100 and continues to 390, as indicated by the arrows. The greatest diameter is between sections 60-200 which corresponds with the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

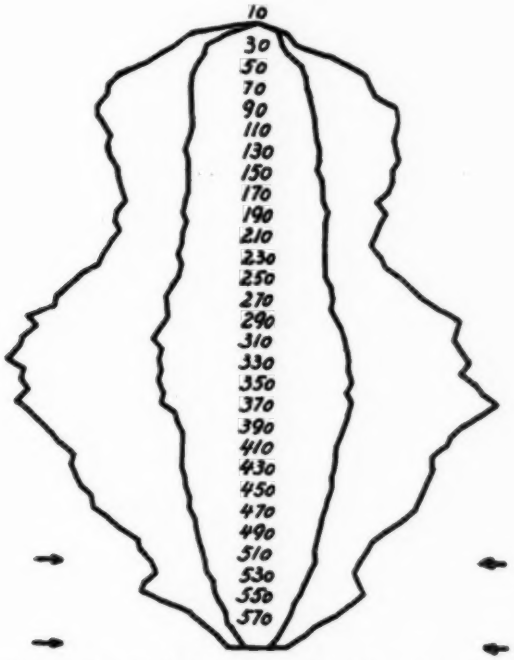

Fig. 9.—Vertical section of brain of cat. Graphic representation of the perimeters of the visible surface, without the medium longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.14 : 1. The cerebellum makes its appearance at 510 and continues beyond the end of the cortex, as indicated by the arrows. The greatest increase in diameters is between section 230 and 410, which corresponds with the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

The brains of two sheep were measured; the total surface was 222 sq. cm. in the horizontal series, giving a ratio of total to visible surface of 2.8:1, and 209.23 sq. cm. in the vertical series, giving a ratio of total visible surface of 2.88:1.

Fox.—One female fox was used. The animal when brought to us was in an extreme state of emaciation. The brain was cut horizontally and yielded forty-seven sections.

As observed from table 1 and figure 2, this brain lost 40 per cent in weight, 38.4 per cent in volume and 25.6 per cent in surface. As seen from these figures,

TABLE 6.—*Perimeters and Ratios of the Visible and Total Surfaces of Sheep*

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
Horizontal—52 Sections													
842.45	977.39	16.39	18.79	35.18	308.34	342.13	5.93	6.57	12.50	2.76	2.86	2.81	59.58
Vertical—92 Sections													
1131.90	1090.29	12.30	13.29	25.59	404.86	413.30	4.40	4.48	8.88	2.80	2.96	2.88	53.23

TABLE 7.—*Visible and Total Surface Areas with and without Shrinkage in Sheep*

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
Horizontal													
27.30	32.28	59.58	78.30	89.30	167.60	32.50	36.20	43.18	79.38	103.80	118.30	222.10	2.80:1
Vertical													
26.40	26.83	53.23	73.85	79.39	153.24	36.5	36.05	36.61	72.66	100.80	108.43	209.23	2.88:1

the loss in volume approximates the loss in weight. The loss in surface, 25.6 per cent, is one fourth of the original weight of the brain (figs. 3, 4 and 5).

Table 8 shows the visible, total and the averages of the perimeters of the right and left hemispheres, the ratio of the total and visible perimeters and the visible surface areas.

Table 9 shows the visible and total surface areas with and without shrinkage, the percentage loss in surface due to shrinkage and ratios of the total to the visible surfaces.

Figure 12 is a representation of the relationship of the visible to the total perimeters.

The brain of one fox was measured; the total surface was 74.04 sq. cm.; giving a ratio of 2.45:1.

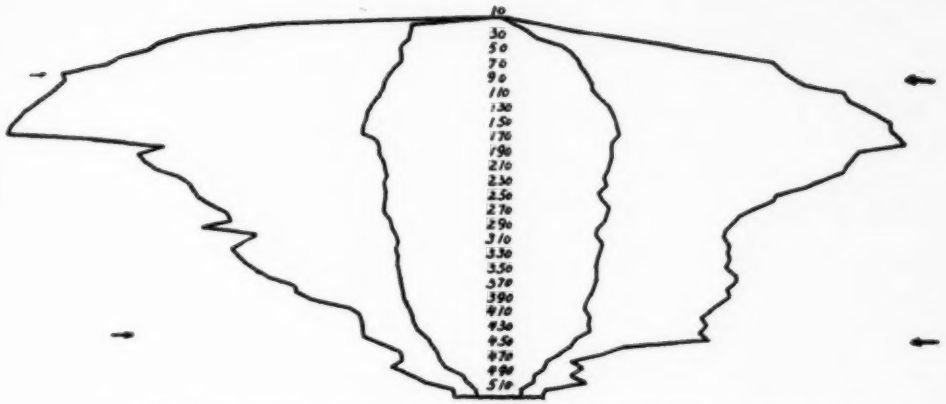

Fig. 10.—Horizontal section of brain of sheep. Graphic representation of the perimeters of the visible surface, without the median longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.8 : 1. The cerebellum makes its appearance at section 80 and disappears at 440, as indicated by the arrows. The greatest increase in diameters is between sections 40 and 220, which corresponds to the presence of the temporal lobes and the greatest area of fissuration. At 260 the fissures become smaller and less numerous and the mid-brain structures increase in size, at the expense of the cerebrum, continuing to section 450. The gaps in the different areas are due to the normal variations in fissuration.

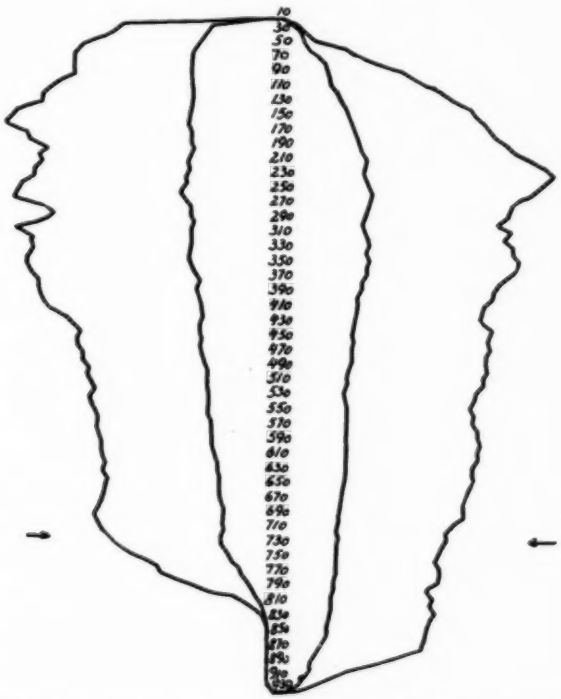

Fig. 11.—Vertical section of brain of sheep. Graphic representation of the perimeters of the visible surface, without the median longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.88 : 1. This brain was cut unevenly, as readily seen from the graph. At the top, the sections on the right were smaller than on the left, while at the bottom there were no sections on the left side. The cerebellum makes its appearance at section 720 and continues beyond the end of the cortex, as indicated by the arrows. The greatest increase in diameter is between section 120 and 420 which corresponds with the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to normal variations in fissuration.

Dog.—One male dog was used. The brain was cut horizontally and yielded sixty-eight sections.

As observed from table 1 and figure 2, this brain lost 48 per cent in weight, 44 per cent in volume and 29.33 per cent in surface. As seen from these figures, the loss in volume approximates the loss in weight, while the loss in surface, 29.33 per cent, is a little less than one third of the loss in weight (figs. 3, 4 and 5).

Table 10 shows the visible, total and the average perimeters of the right and left hemisphere, the ratios of the total and visible perimeters and the visible surface areas.

Table 11 shows the visible and total surface areas with and without shrinkage, the percentage loss in surface due to shrinkage and the ratio of the total to the visible surfaces.

Figure 13 is a representation of the relationship of the visible to the total perimeter.

TABLE 8.—*Perimeters and Ratios of the Visible and Total Surfaces of Fox*

Horizontal—47 Sections

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
490.99	508.79	10.43	10.82	21.25	195.14	213.52	4.16	4.53	8.69	2.52	2.39	2.45	24.06

TABLE 9.—*Visible and Total Surface Areas with and without Shrinkage in Fox*

Horizontal

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
11.51	12.55	24.06	28.92	30.02	58.94	25.60	14.46	15.77	30.28	36.32	37.72	74.04	2.45:1

The brain of a dog was measured; the total surface was 206.88 sq. cm., giving a ratio of the total to the visible surface of 3.28:1.

Monkey.—Two South American long-tailed monkeys were used; the brain of the female was cut horizontally, and the brain of the male was cut vertically. The brain cut horizontally yielded fifty-eight sections, and the one cut vertically yielded ninety-one sections.

As observed from table 1 and figure 2, the two cerebrums have lost 38.4 and 39 per cent in weight, 27.4 and 36.3 per cent in volume and 19 per cent in surface. As seen from these figures, the loss in volume was much less than the loss in weight; the loss in surface, 19 per cent, was about one fifth of the original weight (figs. 3, 4 and 5).

Table 12 shows the visible, total and average perimeters of the right and left hemispheres, the ratios of the total and visible perimeters and the visible surface areas.

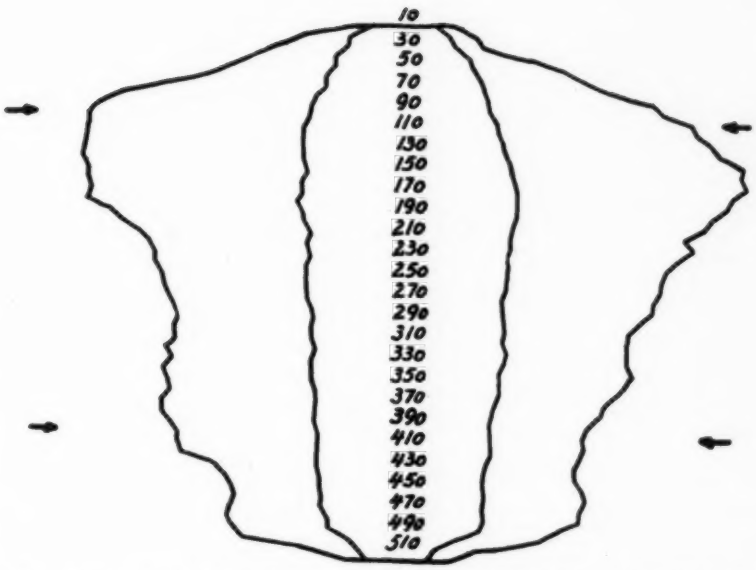

Fig. 12.—Horizontal section of brain of fox. Graphic representation of the perimeters of the visible surfaces without the median longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 2.45 : 1. The cerebellum makes its appearance at section 100 and continues to 400, as indicated by the arrows. The greatest diameter is between section 70 and 230, which corresponds to the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

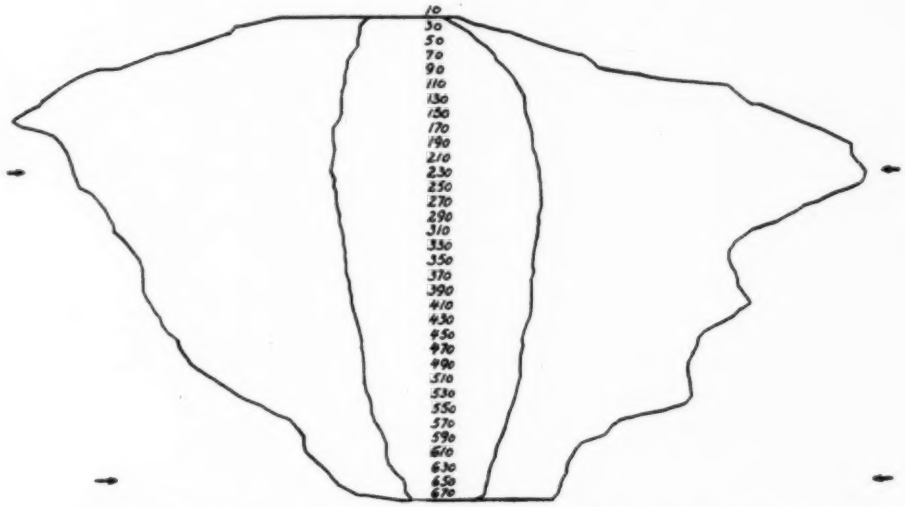

Fig. 13.—Horizontal section of brain of dog. Graphic representation of the perimeters of the visible surfaces without the median longitudinal fissures, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface in this case is 3.28 : 1. The sections were cut slightly uneven, the sections at the top on the right being slightly smaller than on the left. The cerebellum makes its appearance at 100 and disappears at 400. The greatest diameter is between sections 110 and 310, which corresponds to the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

Table 13 shows the visible and total surface areas with and without shrinkage, the percentage loss in surface due to shrinkage and the ratios of the total and visible surfaces.

As is readily seen, the visible surface of the brain cut horizontally is greater than that of the one cut vertically. The total surface, on the contrary, is less in the horizontal series than in the vertical. The ratios of the total to the visible areas of the brain cut vertically are much less than those of the one cut horizontally. The only explanation for this discrepancy is that the monkey whose brain was cut in vertical series was younger than the one whose brain was cut horizontally.

Figures 14 and 15 are a representation of the relationship of the visible and total perimeters.

TABLE 10.—*Perimeters and Ratios of the Visible and Total Surfaces of Dog*

Horizontal—68 Sections

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
1152.67	1205.91	16.95	17.71	34.66	359.28	364.40	5.28	5.36	10.64	3.21	3.35	3.28	48.71

TABLE 11.—*Visible and Total Surface Areas with and without Shrinkage in Dog*

Horizontal

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
24.18	24.53	48.71	76.10	83.90	160.00	29.33	31.26	31.71	63.01	98.40	108.48	206.88	3.28:1

Two brains were cut, one horizontal and one vertical; the total surface was 150.3 sq. cm. in the horizontal series, giving a ratio of total to visible surface of 3.03:1, and 133.19 sq. cm. in the vertical series, giving a ratio of total to visible surface of 2.33:1.

Child.—The brain of a full-term, stillborn girl was cut horizontally and yielded fifty-five sections. As observed from table 1 and figure 2, this brain lost 46 per cent in weight, 42 per cent in volume and 31 per cent in surface. The loss in volume approximates the loss in weight, while the loss in surface, 31 per cent, is about one third of the original weight (figs. 3, 4 and 5).

Table 14 shows the visible, total and the averages of the perimeters of the right and left hemispheres, the ratio of the total and visible perimeters and the visible surface areas.

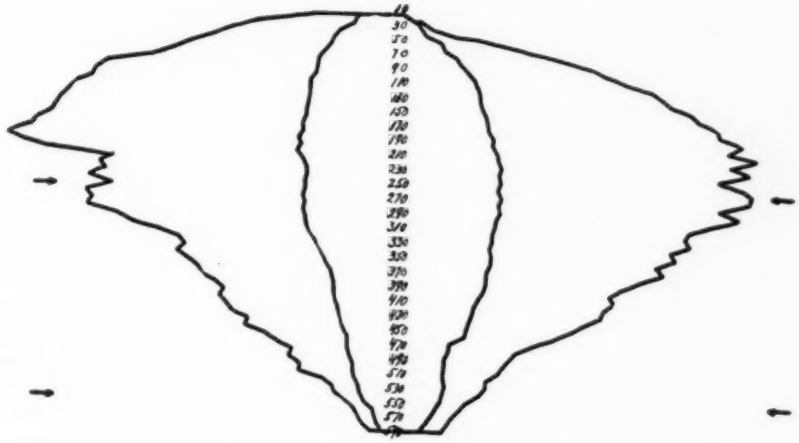

Fig. 14.—Horizontal section of brain of monkey. Graphic representation of the perimeters of the visible surface without the median longitudinal fissure, and the total surfaces of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface is 3.03 : 1. The cerebellum makes its appearance at 250 and continues to 540, as indicated by the arrows. The greatest diameter is between sections 110 and 310, which corresponds to the presence of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

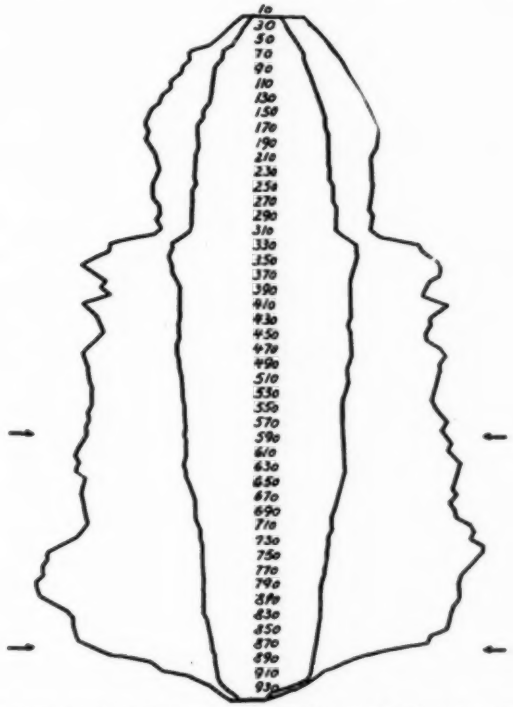

Fig. 15.—Vertical section of brain of monkey. Graphic representation of the perimeters of the visible surfaces without the medical longitudinal fissure, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface is 2.33 : 1. The cerebellum makes its appearance at 580 and continues to 880, as indicated by the arrows. The greatest increase in diameter is between sections 330 and 870 due to the appearance of the temporal lobes and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

Table 15 shows the visible and total surface areas with and without shrinkage, the percentage loss in surface due to shrinkage and the ratios of the total and visible surfaces.

Figure 16 shows the relationship of the visible and total perimeters.

One child's brain was measured; the total surface was 995.46 sq. cm., giving a ratio of 4.08:1.

COMMENTS

As observed from the foregoing experiments, most of the brains, through a process of shrinkage, lost from 38 to 63 per cent in weight,

TABLE 12.—*Perimeters and Ratios of the Visible and Total Surfaces of Monkey*

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
Horizontal—58 Sections													
843.23	847.41	14.50	14.60	29.10	278.97	277.12	4.79	4.78	9.57	3.02	3.05	3.03	41.52
Vertical—91 Sections													
917.17	920.95	10.07	10.21	20.38	404.47	414.47	4.44	4.55	3.99	2.27	2.25	2.26	49.53

TABLE 13.—*Visible and Total Surface Areas with and without Shrinkage in Monkey*

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
Horizontal													
20.90	20.62	41.52	63.00	63.30	126.30	19.00	24.87	24.55	49.42	74.96	75.34	150.30	3.03:1
Vertical													
24.45	25.08	49.53	55.50	56.44	111.94	19.00	27.10	29.85	58.08	66.06	67.14	133.19	2.33:1

and from 27 to 54 per cent in volume. The great loss in weight and volume depended on the chemical constituents and the amount of water in the brains. Evidently the brains of lower forms contain a greater quantity of water. The percentage loss in surface is about the same in most of these animals, with the exception of the two monkeys. In the percentage loss of weight, volume and surface, the child's brain approximates the rest of the animal series with the exception of the monkeys, due to the fact that this child was stillborn and the brain contained large quantities of water.

The relationship of the visible and total surfaces of the brains of the same animal cut in two dimensions was about the same, with the

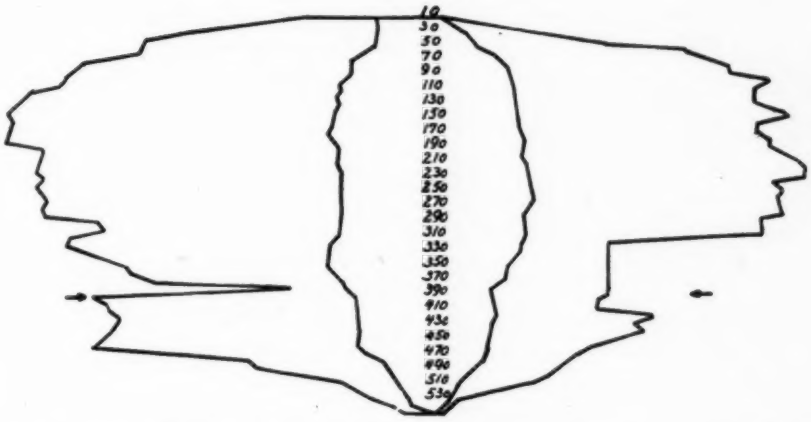

Fig. 16.—Horizontal section of brain of child. Graphic representation of the perimeters of the visible surface without the median longitudinal fissures, and the total surface of each cortical section. The small area in the center is the visible surface. The ratio of the visible to the total surface is 4.08: 1. The two great gaps are due to the cutting of the brain in two halves. The cerebellum makes its appearance at section 390 and continues beyond the end of the cortex, as indicated by the arrows. The greatest diameter is between sections 80 and 300, which corresponds to the presence of the temporal lobe and the greatest area of fissuration. The gaps in the different areas are due to the normal variations in fissuration.

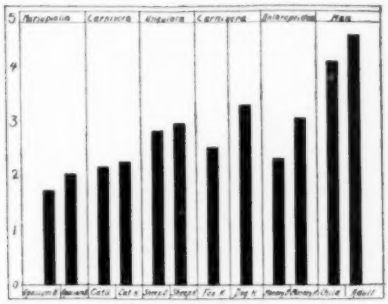

Fig. 17.—Graphic representation of the ratio of the total to visible surfaces in the vertebrate brains measured. The black columns indicate how many times the total surface is larger than that of the visible surface.

exceptions of the opossums and the monkeys. The brain of the opossum cut vertically (table 3) showed a smaller ratio between the visible and the total surfaces than the one cut horizontally. The explanation was that this animal was younger, with a body weight and a brain weight less than the other. The brain of the monkey cut vertically showed a smaller ratio between the visible and the total surface. Although this brain weighed as much as the other, the animal appeared much younger. This may serve as an explanation for the smaller ratio.

The dog's brain showed a higher relationship between the total and visible surfaces than that of the monkey. As the dog is a highly

TABLE 14.—*Perimeters and Ratios of the Visible and Total Surfaces of Child*

Horizontal—55 Sections

Total Perimeter		Average Total Perimeter		Average Total for Right and Left, Cm.	Visible Perimeter		Average Visible Perimeter		Average Total Perimeter for Right and Left, Cm.	Ratios of Perimeters of Total and Visible Surfaces			Visible Surface Without Shrinkage, Sq. Cm.
Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right, Cm.	Left, Cm.	Right, Cm.	Left, Cm.		Right	Left	Both	
2050.02	2313.45	37.27	42.06	79.33	493.69	569.93	9.14	10.36	19.50	4.07	4.06	4.068	185.99

TABLE 15.—*Visible and Total Surface Areas with and without Shrinkage in Child*

Horizontal

Surface Without Shrinkage						Shrinkage, Percentage Loss	Surface With Shrinkage						Ratio of Total to Visible Surfaces
Visible Surface			Total Surface				Visible Surface			Total Surface			
Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.		Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	Right, Sq. Cm.	Left, Sq. Cm.	Both, Sq. Cm.	
87.50	98.50	185.99	357.45	398.19	756.64	31	114.60	129.00	243.60	468.53	524.49	995.46	4.08:1

domesticated animal and the cebus is a low form of monkey, it is not at all surprising that the brain of the dog should have a greater surface area.

Definite conclusions from these measurements cannot be drawn, as one would have to know more about these animals, i.e., their exact age, their behavior and measurements of a greater number of brains of the same species.

SUMMARY

An attempt was made to give an idea of the measurements and relationships of the cerebral surfaces of various animals. While definite conclusions could not be drawn from the small numbers of brains measured, it is safe to state that the total surfaces as well as the relationship of the total to the visible surfaces increase as one ascends the animal scale and reach their acme in man.

BRIEF ATTACKS OF MANIC-DEPRESSIVE DEPRESSION *

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Although descriptions of manic-depressive depression are numerous, there are few reports of a form of the disease which is common and significant. I refer to attacks lasting from a few hours to a few days. The duration of attacks of manic-depressive depression as presented in the literature is usually from a few weeks to several months and even years. Strohmayer,[1] however, reported that such disturbances may last for from a few hours to a few days. Stransky,[2] Kraepelin,[3] White [4] and Rosanoff [5] gave the minimal duration as a few days, but none of these writers considers such fleeting attacks as common or important. Bleuler [6] said that "by way of rare exception individual patients have a tendency to complete their attacks in one or two weeks." In 1901, in a study of patients with cardiac, pulmonary and abdominal disease, Head [7] described attacks of morbid depression lasting an hour or more and recurring at frequent intervals throughout a day or two and attributed the mental change to reflex visceral pain. However, he stated that such attacks may occur as a primary mental manifestation.

So far as I have been able to ascertain, Gregory [8] was the only writer who appreciated the frequency and importance of these short attacks of primary mental depression. He even expressed the belief that they were more frequent than the longer and more severe attacks requiring hospital care. He also discussed their frequent relation to

* Submitted for publication, April 30, 1928.

* Read at a meeting of the Chicago Neurological Society, April 19, 1928.

1. Strohmayer, W.: Manisch-depressive Irresein, Wiesbaden, Bergman, 1914.

2. Stransky, E.: Lehrbuch der allgemeinen und speziellen Psychiatrie, Leipzig, F. C. W. Vogel, 1914.

3. Kraepelin, E.: Manic-Depressive Insanity and Paranoia, translated by Mary Barclay from the Eighth German Edition of the Text-Book of Psychiatry, vols. III and IV, Edinburgh, E. and S. Livingstone, 1921.

4. White, W. A.: Outlines of Psychiatry, Washington, Nervous and Mental Disease Publishing Co., 1926.

5. Rosanoff, A. J.: Manual of Psychiatry, New York, John Wiley and Sons, 1927.

6. Bleuler, E.: Textbook of Psychiatry, translated by A. A. Brill, New York, The Macmillan Co., 1924.

7. Head, Henry: Certain Mental Changes that Accompany Visceral Disease, Brain **24**:346, 1901.

8. Gregory, M. S.: Transient Attacks of Manic-Depressive Insanity, M. Rec. **88**:1040, 1915.

attacks of alcoholism (inebriety) and their misinterpretation as some other form of mental disorder or as some somatic disturbance. Of more than fifty articles pertaining to manic-depressive psychoses published in journals during the last fifteen years, only one other author [9] was found to consider these brief attacks, and he did so only casually.

It seems reasonable to conclude that these brief and attenuated attacks of manic-depressive depression have received such scant attention in the literature because practically all of the books and most of the periodic writings on mental disorders are produced by men working in hospitals or sanatoriums for the insane. These men do not see the dwarf attacks and know of them only from the anamnesis. For them, such attacks are rare and unimportant. On the other hand, Gregory's experience was in the psychopathic ward of a huge municipal hospital, largely fed by the police department and free dispensaries; this source of supply and material is comparable to private practice. But the man who sees most of these patients, when any one sees them, is the family physician or the neurologic consultant. Consequently, it seemed worth while to present the subject from the standpoint of private practice.

In a study of the histories of 633 cases of recurrent attacks of manic-depressive depression in the private records of Dr. Hugh T. Patrick, I found eighty-eight cases, or 13.9 per cent, with a history of attacks lasting from a few hours to a few days. Probably this figure understates their actual frequency, as doubtless not all patients were questioned on this point. Although the histories studied had been taken during a period of thirty-two years, 83 per cent of the cases in which the brief attacks had occurred were found in the records of the last ten years.

I believe that such diminutive attacks of manic-depressive depression are rarely recognized for what they are. As these attacks of primary depression are frequently accompanied by a feeling of weakness, ready fatigue, head pressure, constipation, anorexia, coated tongue, epigastric or subensiform distress, eructations, flatulence, perhaps regurgitation or vomiting and palpitation, the patient and physician alike are apt to conclude that the trouble is somatic. Not finding organic disease, the physician may predicate neurasthenia, hypochondria, hysteria or nonsense. Then, as following a brisk cathartic or mere reassurance, the patient promtly recovers—perhaps before the cathartic had time to work—the erroneous diagnosis is confirmed. As a result, in the next attack the same wrong diagnosis and treatment are repeated.

Is this depression of from a few hours to a few days really morbid or merely a normal fluctuation in mood as a reaction to temporary experiences? If morbid, is it primary depression or excessive reaction

9. Clark, S. N.: The Makeup of Atypical Cases of Manic-Depressive Insanity, J. Nerv. & Ment. Dis. **45**:424, 1917.

of sensitive persons to the ordinary ills of a troublesome world? I am convinced that these miniature attacks are the disorder recognized as manic-depressive depression. The symptoms are exactly like those of longer attacks: profound sadness and unhappiness without obvious cause, self reproach, self blame, self derogation, lack of initiative, lack of response in usual interests and keen awareness of this lack, avoidance of friends, a feeling of hopelessness, death wishes, and inclinations or desire to commit suicide; there is an absence of the phobias and other symptoms of psychasthenia with preservation of intelligence and no lack of emotional response.

The suddenness and apparent causelessness of their appearance and disappearance are characteristic of these brief attacks. Careful analysis shows that these attacks are not brought on by causes such as deaths or illnesses of dear ones, domestic difficulty, financial losses, frustrated hopes, unrealized or unrealizable desires, quarrels, reproaches, loneliness and "overwork." The well known antidotes for depression such as a philosophic outlook, the company of friends, amusements, diversions, rest, change of scene and good news, do not cause the attacks to disappear. Instead, one finds a person in a normal mood who, without apparent cause, becomes within a brief period profoundly sad and unhappy; in spite of all attempts to cheer him, the attack remains for from a few hours to a few days; when it does disappear, it does so as abruptly and mysteriously as it came.

That the brief and the long attacks are the same disorder is further indicated by their occurrence in the same person, the only essential difference being the time factor. In fifty-one cases the brief attacks preceded longer ones, sometimes by months, sometimes by years, not rarely by decades. In twenty-eight cases the transient attacks appeared after or between more protracted ones. In nine cases there were brief attacks only; whether these patients will later be affected with longer attacks none can tell.

The following brief citations will serve to illustrate. Group 1 presents cases in which long attacks did not occur, the entire illness running its course in periods of depressions of from a few hours to a few days. In group 2, the brief attacks preceded the long ones. In Group 3, the transitory attacks appeared after one or more protracted ones.

REPORT OF CASES

GROUP 1: CASES IN WHICH THE DISORDER RAN ITS COURSE IN BRIEF ATTACKS ONLY

CASE 1.—A saleswoman, aged 23, stated that the father had been quick tempered, domineering, and had had "blue spells"; the mother was easily excited, "highstrung," and had severe migraine; one sister was "nervous" and "highstrung"; a paternal uncle, aged 80, had a "chronic grouch" and had had periods of depression

during the last twenty years. Since the age of 13 or 14, for a week or ten days preceding menstruation, the patient had been unhappy, sad, had thought slowly, had had less initiative and interest, occasional crying spells and death wishes, and had contemplated suicide. During such periods she awoke feeling that she was not alert, that she could not handle customary situations and was in a state of inertia which lasted the entire day. During such episodes she felt that she was "no good," that she could not depend on herself, and she did not care to talk to any one because she felt that her voice was not right. Such attacks also occurred spontaneously between the menstrual periods, but then lasted only part of a day, usually during the morning. In the periods of depression she always blamed herself; she was not resentful and did not criticize others. She also had periods of from one to two days in which she felt "all pepped up" and overenthusiastic; she felt "as if she owned the world." From this overreaction she sometimes went into a "blue spell," but usually not. For the past four years she had occasionally been troubled by a feeling of abdominal distention and cardiac palpitation which became aggravated in the depressed periods, especially for one or two days before menstruation.

Case 2.—A woman, aged 30, with unimportant family and personal history, had been confined eight weeks before she was seen. Labor was normal. During the last two months of pregnancy she awakened during the night and remained awake an hour or more, feeling discouraged "because she could not sleep." Two weeks after confinement the milk disappeared and she became discouraged, but not for long. Three weeks after the birth of the infant she began to have crying spells that she could not explain, and afterward, without obvious cause, she had attacks of depression which lasted a number of hours every day with the exception of two or three days. During the periods of depression, she "doesn't care for anybody or anything;" it is "difficult to make herself do things;" "work seems very difficult although before it was easy." During these periods she had difficulty in making decisions, found it hard to concentrate, did not show any response to her usual interests, took a long time to dress, had to be urged to eat, and felt that she was neglecting her child. Between the attacks, she felt normal for several hours. During the periods of well being, she thought of the depression, felt that it was foolish, was worried about it, but did not understand it.

Case 3.—A minister, aged 43, whose family history was without significance except that his mother had been a nervous invalid for years and was depressed most of the time, had always been "nervous," "highstrung" and "worrisome." Since about the age of 23, he had had periods of depression lasting from two to seven days and recurring about five or six times yearly, usually, he thought, after hard mental work. During these attacks he was sad, unhappy, had an aversion for people, poor appetite, crying spells, fear of high places and fear of shaving because he might harm himself, and contemplated suicide.

Case 4.—A married woman, aged 41, had seven paternal uncles all of whom were more or less depressed and one of whom was a confirmed melancholic. The mother was of a "nervous" type; the patient had always been "nervous" but was better after the age of 30, when her first child was born. She also had migrainous headaches, and was subject to what she called "nervous jags." With the menses, and sometimes between them, she had either a headache or a "jag." These "jags" lasted from one hour to three days, generally not more than one day. During these attacks, she talked continuously and walked the floor. With the later upsets she contemplated suicide and thought that her life was an entire failure and that she was of no help to her husband and children. The longest free interval was

about two years, the shortest about one week. In her latest attacks, she showed a tendency to blame others rather than herself. These attacks came and went without obvious or reasonable cause.

CASE 5.—A farmer, aged 46, with unimportant family history, had had a chancre at 22, but the Wassermann reaction of the blood and of the spinal fluid was negative when he was examined. Eight months before he had begun to have attacks, lasting from a few days to a week, in which he had "bloating spells" after eating and felt pain and distention in the abdomen until he "broke wind." At the same time he was sad, unhappy and discouraged and feared sudden death, paralysis and insanity. During these attacks he slept poorly, awakened at 4 or 5 a. m. and in these early hours was more despondent than ever; he tired easily, had loss of "ambition," found it hard to concentrate, and complained of poor memory. The attacks occurred without ascertainable cause and started and terminated abruptly. Between them, for from several days to three or four weeks, he was cheerful and ambitious; appetite and digestion were good, and his thoughts during the attack seemed silly to him. He was seen during an attack of depression which had lasted four days and presented a picture typical of primary depression.

CASE 6.—A married woman, aged 45, with unimportant family history, twenty-one years before examination underwent an induced abortion. She had been in bed for one week, and during this time she had been depressed and had had crying spells. A year later, she had a spontaneous abortion, was sick two weeks, bled a great deal, lost weight, was "blue" and discouraged and cried a great deal. Since then, without apparent cause, she had had periods of depression, starting suddenly, lasting from one to four days, and ending abruptly. During these attacks she was profoundly sad and unhappy, wept, slept poorly, wished for death and found it difficult to concentrate; on a few occasions she had become confused in familiar surroundings. At times she also complained of a feeling of pressure in the head and of cardiac palpitation. She stated that the depression was somewhat relieved by working, company, visiting, and amusements, but this seemed doubtful.

CASE 7.—A clerical worker, single, aged 22, whose father was excitable and quick tempered, and whose mother was of a quiet, timid type, always had been "moody;" he was either optimistic, alert and quick or he thought "slowly" and was pessimistic. The latter mood was more frequent than the former; it usually lasted about one and one-half days, began and ended abruptly, and returned again in a few days. These moods were most often spontaneous, but he thought they might have been aggravated by excitement or irritation. For the preceding eight months, he had been more depressed than usual with fewer good days. During the attacks he was sad, unhappy, pessimistic, felt "tired," wanted to lie down, feared that he would lose his position and his friends, wished for death, and slept fitfully for only five or seven hours.

CASE 8.—A wholesale grocer, aged 36, whose father was "nervous" and had had a breakdown with depression between the ages of 45 and 48, whose mother died at 56 of diabetes, and whose sister was of a "nervous type," had always been "nervous," "highstrung," "worrisome" and inclined to have "blues" lasting from one to two days and recurring at irregular intervals. For one and a half years previous to examination, he had been growing progressively more "nervous" and irritable and became depressed more frequently, the periods of depression lasting three or four days. During the attacks he was sad, unhappy, avoided people, felt as if he would like to get away from everything and everybody, as though he would rather be dead, feared that he might become insane, wept a great deal and slept poorly;

he had no resentment toward, nor criticism of others. Gastric symptoms were also present in the form of belching, coated tongue, nausea and vomiting. The attacks came and departed suddenly without ascertainable cause.

Case 9.—An architect's wife, aged 34, whose father was "nervous and high-strung," the family history otherwise being of no significance, as an infant was rather sickly; as a child she was probably spoiled and had temper tantrums. Since childhood she had been subject to hay-fever and asthma. At 26, intermittently for one year, she had attacks of "constriction" in the throat like "asthma." For the past twelve years she had been subject to attacks of nausea lasting from two or three days to three or four weeks, with free intervals of several months. She described this "nausea" as an uncomfortable feeling in the substernal region, a feeling "like a scare" that one would get when speaking in public. One year previously a gastro-intestinal examination, with roentgenograms, was made and gave negative results. When the patient was seen, these attacks were the sole complaint. Questioning, however, elicited all the reactions of manic-depressive depression except the desire to commit suicide. In this attack and in the others she was profoundly unhappy and sad without obvious cause, reproached and blamed herself, lacked initiative and energy, had difficulty in making decisions, was not responsive to usual interests, avoided friends, slept poorly, and had no resentment toward, or criticism of, others. It is interesting to note that when she entered the examining room, she did not have epigastric distress, but when she heard the opinion of her case she was discouraged and began to feel this discomfort at once.

GROUP 2: CASES IN WHICH BRIEF ATTACKS PRECEDED LONG ONES

Case 10.—A married woman, aged 35, whose mother had had several attacks of depression lasting several months, was always "nervous," "worrisome" and inclined to be depressed much of the time. At 29, she began to have periods of more marked depression lasting from one to three days, during which she was profoundly sad and unhappy, kept to herself, did not talk, wept considerably and slept poorly. The attacks came on and disappeared suddenly without obvious cause. At first these periods occurred every two or three months; after the age of 33 they came on about once a month. When first seen she had been continuously depressed for six weeks. This attack started with worry over a sick sister; three weeks later the sister died, and the patient became much worse. She was profoundly unhappy and dejected, had marked insomnia and terrifying dreams, wept most of the time, was afraid to be left alone, yet kept to herself, and contemplated suicide. Her head felt "full," and when she attempted to concentrate her "mind became a blank"; she was afraid that she was becoming insane. She blamed no one for her trouble, did not feel resentment and was not critical of others. She also complained of a more or less constant feeling of pressure over the vertex, poor appetite and constipation. Evidently she ate poorly as she had lost 10 pounds (4.5 Kg.) during the last three weeks of the illness.

Case 11.—A married woman, aged 41, had always been "nervous," "highstrung," hypersensitive, easily offended and easily depressed. The father had been of a "nervous" type and had died of diabetes. Three sisters and a brother were "nervous;" the mother and one sister had migraine. The patient was always more nervous and depressed three or four days before the menses, and complained of headache, vomited, wept and had insomnia. At 32 or 33, she began to have, at irregular intervals, periods of three or four days in which she was sad, unhappy and worried, and in which she wept and had insomnia. At 36, these episodes

became more frequent and lasted longer. These attacks appeared and disappeared suddenly and were not due to any ascertainable cause. At 38, she went into an attack of depression which lasted three years and ten months, although the course of the illness had been marked by fluctuations. Again she was sad, dejected, worried without obvious cause, wept frequently, had difficulty in falling asleep, avoided friends and relatives, did not care to go out, had suicidal intentions and feared that she was going insane.

CASE 12.—A farmer's wife, aged 52, had passed the menopause at 45. The mother and one sister had had periodic headaches. She was always "nervous," "highstrung" and "worrisome." At 35, she had typhoid fever, and after that she had "blue spells" lasting from one to two days about two or three times a year. As she grew older, the duration of the spells increased to five or ten days. During these spells she suddenly and without apparent reason became sad, unhappy, felt inappetent, weak, had loss of energy, and lost interest in her usual pursuits. After a few days, the attack terminated suddenly. When seen she was in a period of depression which had lasted three months. Again without ascertainable cause she was sad, unhappy, weak, easily fatigued, ate poorly, had lost 28 pounds (12.7 Kg.), had self-derogatory ideas, thought life was not worth living, and a month before had attempted suicide by cutting her throat.

CASE 13.—A nurse, aged 33, with unimportant family history, as a child had always been timid and "afraid of people." From infancy to about 13 she had had attacks of slight headache with nausea and vomiting, lasting about from one to three days and occurring about two or three times yearly. Since childhood she had had "blue spells" lasting from one to four days and occurring without apparent cause two or three times a year. These blue spells had begun and had ended suddenly. When seen she was in an unended attack of depression of three months' duration. This last attack was just like the "blue spells" except that it lasted longer and she felt weaker. Without apparent cause she was sad, unhappy, discouraged, had a feeling of hopelessness and worthlessness, felt that she was "no good," wept a great deal, felt that she was friendless, lost all energy and enthusiasm, and had death wishes. She thought that she was being belittled by people, but had no definite ideas of persecution. She continued working as a school nurse throughout the illness.

CASE 14.—A married woman, aged 53, since childhood and until the age of 51 had been subject to severe headaches with vomiting. From 46 to 51 she passed through the climacteric with excessive hot flushes which persisted until seven months before she was seen. She was always "nervous," easily excited, easily brought to tears, religious and energetic. Since childhood she had had periods of mild depression lasting from one to three days and occurring spontaneously about two or three times yearly. During such states she suddenly became sad, unhappy, had a feeling of discouragement and death wishes, but never attempted suicide; she wept occasionally; there was marked change from her usual enthusiastic and hyperkinetic behavior to a state of quiet sadness. These attacks ended suddenly. When seen, she had been depressed for seven months. She thought the attacks had begun with constipation, "gas" and pain in the abdomen. Then she became restless, slept badly and feared cancer. A month after the onset she had a distinct feeling of hopelessness; she expressed at times a fear of and at other times a desire for death. She grew gradually more depressed, with marked crying spells, marked despair, and in the last month refused at times to go out and to see people.

CASE 15.—A merchant, aged 45, whose father had had "melancholia" and whose brother was of a "nervous" type, had always been "nervous" and "worrisome."

At 20 (twenty-five years before examination) he suddenly had become sad and unhappy, and frequently had wept. This condition lasted about a week, then gradually cleared up and was considered an attack of "heat prostration." At 27, during another heat wave, he suddenly again became profoundly depressed and wept frequently; this lasted for several days (heat prostration?). When seen, he was in a state of depression of three weeks' duration. Without apparent cause he was profoundly sad and unhappy, wept frequently, slept poorly, had no "ambition" to do anything and did not desire to see any one; he reproached himself severely for occurrences of twenty years before. He could not attend to business and wanted to die.

Case 16.—A happily married woman, aged 35, whose family history was without significance except that her father had had migraine and two of her four children were inclined to be "nervous" and "highstrung," and whose early history was unimportant, at 20 began to have attacks, occurring at variable intervals, in which she suddenly became depressed and remained so for a day or two. She thought that they often were precipitated by worry, such as illness in the family. During these periods she was profoundly unhappy, sad, had no initiative, lost interest in her usual pursuits, did not care to see any one, was "disgusted" with herself, felt a loss of self confidence, did not desire to do anything, spent most of the time lying down, and wished that she were dead. The depression apparently was relieved by "vomiting," but was liable to return again when food was taken. From 20 to 26, she had only brief attacks. At 26 she had a similar attack lasting two or three months. After each of her pregnancies she had similar attacks lasting two or three months, and it is noteworthy that during pregnancy she was entirely free from depression. In the two years prior to examination, she had three attacks lasting one day, which occurred several months apart. When first seen she had been depressed for six days. She was again sad, unhappy, had marked loss of initiative and energy, was self-derogatory, thought she was not a good wife and mother, found it difficult to make decisions, slept badly, was "nauseated" and "vomited"; she ate poorly because she was afraid of "vomiting" and thought that she lost weight. This attack terminated five days later. In the month following she had similar periods of depression about once a week lasting from one to three days, but with these attacks she did not vomit. The "vomiting" in this case was factitious. She had an uncomfortable feeling in the epigastrium, or slightly above, which she attributed to the stomach and by voluntary efforts regurgitated food. As the years went by this regurgitation grew more and more easy and complete, and at the same time the idea that her trouble came from the stomach grew. Although she stated that vomiting relieved the depression, some attacks lasted two or three months although she vomited frequently. Various diagnoses were made in this case; one competent internist considered it migraine without headache, notwithstanding the fact that some of the attacks lasted two or three months, which does not occur in migraine.

Many patients with manic-depressive depression have a peculiar feeling in the epigastrium or pit of the stomach obviously due to the depression. Although this disagreeable sensation fluctuates, it is practically constant, and many patients not unreasonably refer it to the stomach. Apparently, this sensation is due entirely to the emotional state and may be compared to the momentary pang felt in the same region which many people have in witnessing an accident or some unexpected incident. Or it might be compared to the sensation, often quite intense, which children have in this region when sobbing or under intense disagreeable emotions. One small sensitive boy objected to having reread to him a sad story because, he said, it made him feel so bad "here," placing his hand below the ensiform cartilage.

CASE 17.—A bank cashier, aged 57, whose half-sister had had an attack of hypomania and whose brother had had an attack of depression lasting two years, had always been steady, industrious, conscientious and sociable. For many years, until he was 42, he had had attacks three or four times yearly which lasted from one to two days during which suddenly and without obvious cause he became sad, unhappy, had a diminution of energy, had constipation, a coated tongue and, he said, a sallow complexion. At 42, following financial reverses, he had an attack of depression lasting four months. During this time he had crying spells, a feeling of worthlessness, contemplated suicide, slept poorly and feared that he would be unable to provide for his family although they were well provided for. After this attack he remained well until nine months before he was seen, when he entered another period of typical primary depression which had been continuous since.

GROUP 3: CASES IN WHICH BRIEF ATTACKS OCCURRED AFTER OR BETWEEN LONGER ATTACKS

CASE 18.—A merchant, aged 32, whose family and personal histories were without significance, at 24 had an attack in which for two months, without apparent cause, he was profoundly sad, unhappy, had loss of initiative and energy and was taciturn. The appetite was poor, and he lost some weight. Following this, every two or three months, were periods lasting two or three days in which abruptly and without obvious reason he became sad, dejected, had marked diminution in initiative and energy, and talked little. These attacks ended suddenly. They continued until about ten months before the examination. At that time, he had another attack of depression lasting three months. Without ascertainable cause he was again sad, unhappy, lost interest in his usual pursuits, had little initiative, found it difficult to perform his usual tasks, did not care to talk to any one and was not critical of nor resentful toward others. The attack in which he was seen had started two or three weeks before. Without apparent reason he was again profoundly sad and unhappy, had weeping spells, lack of initiative and energy, lack of interest in his daily affairs, spoke little, at times slept poorly, reproached himself only, but denied contemplating suicide. The appetite was poor, and for the last week he had awakened with nausea but had not vomited.

CASE 19.—A philanthropic campaign director, aged 36, whose father had been depressed for two years previous to death and whose mother had been "nervous," had always been sensitive; he had many acquaintances but few friends. At 29, he had an attack lasting three months in which he was profoundly sad and unhappy, had little initiative and energy, avoided his friends and acquaintances, was slow to fall asleep, wept frequently, had poor appetite and lost weight. He thought that this was brought on by differences with co-workers. Following this, at irregular intervals, he had periods lasting from two hours to two days, most often one day, in which suddenly and without apparent cause he became sad, unhappy, slept poorly and had loss of initiative, energy and interest. These attacks ended suddenly. He was seen during an attack which had lasted seven weeks. Without ascertainable reason he was dejected and had weeping spells and difficulty in falling asleep. He avoided people, had lost his self confidence and felt unable to perform his usual tasks; he "hated" himself, felt that he was guilty of bringing this trouble on himself and thought that he would be better off dead.

CASE 20.—A married woman, aged 37, stated that the father was "nervous" and "highstrung" and had migraine, and that she thought that some of the paternal uncles and aunts were subject to depression. Since the age of 13 she had had periodic headaches lasting from one-half to one day; her daughter, aged 13, had

had similar headaches for six months. Otherwise she had been in good health, happy, enthusiastic and sociable, until the age of 32 when she had an attack of depression that lasted six months. After this she had "blue spells" about two or three times yearly. These "spells" came on spontaneously and suddenly, lasted from one-half to one day and ended suddenly. During these attacks she felt sad, unhappy, weak and easily exhausted and wept and had a fear of dying. She was seen in an attack of depression which had started six months before. At the onset she felt fatigued, as if everything were wrong; she felt unable to do the day's work and unable to make decisions and lost interest in her usual pursuits. Later she became more self derogatory, thought that she was a burden to her family and that she was suffering because of her sinfulness. She had frequent weeping spells, ate poorly and lost 7 pounds (3.2 Kg.).

Case 21.—A junior executive, aged 31, whose family history was unimportant except that the father was serious minded, a "worrier" and had had migraine, at 9 had a "blue spell" lasting one month, in which without ascertainable cause he was sad and unhappy, lost initiative and energy, lost interest in his usual pursuits, slept badly and had crying spells. At 13, he had another attack lasting two months. From 16 to 22, he had similar periods lasting one or two days at irregular intervals. At 22 he had an attack lasting one week, followed by short attacks lasting from one to two days at intervals of from six to nine months until he was 27. After this the periods of depression lasted from a few hours to a week. At 28, he had an attack lasting six weeks, which was followed again by the short attacks. When seen, he had an attack which had lasted six weeks. In all of the attacks, long and short, he was, without ascertainable cause, sad, unhappy, had little initiative and energy, lost interest in his usual pursuits, found it hard to make decisions and avoided people. He felt that he was guilty of bringing this trouble on himself, accused himself of being "no good," contemplated suicide, slept badly, wept frequently and always felt worse in the morning.

Case 22.—An electrical engineer, aged 43, with an unimportant family history, at 29 began to have symptoms of gastric ulcer. At 34 he underwent gastroenterostomy; this was followed by gradual improvement of the symptoms. A short time after the symptoms began to improve he was put on a restricted diet and "at once" became depressed and remained so for two years; however, he worked throughout. At 41 he had an attack of depression lasting four or five days. About two and one-half weeks before he was first seen, he again felt epigastric distress; it was not severe, not affected by food, and not accompanied by vomiting. He was taken to a hospital and examined, but nothing abnormal was found except evidences of an old pulmonary tuberculosis. Following the examination, he became depressed and felt constant distress in the epigastrium. This depression lasted about three weeks. Following this, he had attacks every ten or fifteen days which lasted two or three days and during which he was sad, unhappy, avoided people and lost interest in his usual pursuits; his appetite was poor; he had insomnia and felt epigastric distress and a tingling sensation in the forearm and legs. These attacks commenced and ended abruptly, and no cause was found. Between the attacks the epigastric discomfort and anorexia disappeared, and the tingling was improved.

SUMMARY

In eighty-eight of 634 private cases of recurrent periods of manic-depressive depression, there were definite attacks lasting from only a few hours to a few days. These brief attacks may precede or succeed the more extended ones, or they may occur alone.

Although the data supplied by these records show that only 13.9 per cent of patients with manic-depressive depression have the brief attacks, probably the real ratio is much higher. Very likely, most patients subject to brief attacks alone are not seen by physicians or are seen only by general practitioners, since these illnesses are so short, may appear at long intervals and apparently yield to some simple remedy. Consequently they may be repeated for decades without skilled observation until the first protracted depression. Even then the previous minor attacks may be overlooked or ignored. Furthermore, periods of protracted depression may not occur. Recognition is prevented by general ignorance of mild manic-depressive depression, especially of these extremely brief attacks, and because both the patient and the physician focus their attention on attendant or fortuitous somatic disorders or complaints. In this, as in other sickness, the patient is apt to complain not of what he has but of his interpretation of what he has. Consequently the diagnosis is apt to be dyspepsia, indigestion, biliousness, autointoxication, constipation, disordered liver, gastritis, headache, neurasthenia, nervousness, heat prostration, hysteria or the equivalent of migraine.

A correct diagnosis is important in order to permit the institution of appropriate treatment, to allow a reasonable prognosis and, if possible, to prevent suicidal (possibly homicidal) attempts.

An attempt has not been made to consider transient depressions which voice themselves in inebriety or other excesses.

ABSTRACT OF DISCUSSION

DR. CHARLES F. READ: Most of my experience with mental disorders has been in institutions where one sees only cases of long duration. I have seen practically no cases which ran as brief a course as that Dr. Paskind has described. I do not doubt his observations and conclusions, but wish he might give some criteria as to prognosis, as to whether the attacks may increase in duration. I presume that gastro-enterologists take care of many of these patients, and probably suicide ends many cases.

DR. HUGH T. PATRICK: I should like to know whether other men see these brief attacks. They have been of extreme interest to me for several years, having become more frequent in recent years, but I probably overlooked them before. Several years ago, I had a talk with Gregory about them, and he was so enthusiastic that I became more observant. I am convinced that many of the attacks are typical manic-depressive depression. They have all the characteristics of the prolonged attacks. I think that many cases of mild manic-depressive depression are overlooked entirely, and are called by various names among the psychoneuroses. I am more certain of this because frequently, as Dr. Read mentioned, some of the patients commit suicide, and apparently no one knows just why. I am perfectly certain that many of the patients who are never ill enough to get into a sanatorium or institution are sufficiently ill to commit suicide. I have an unpleasant memory of a patient whom I did not consider suicidal at all, but who ended his life under a locomotive a few days after I saw him for the second time. A few such cases

make one more observant and apprehensive. Most of them are miscalled neurasthenia. I have not seen suicide in one of the brief attacks, but am interested to know the experience of others with these cases. A patient who had brief attacks, lasting a day or two on some occasions and only a few hours on others, did not stop work but had an appendectomy done because of the peculiar sensations due to the manic-depressive depression. Those signs in the epigastric region, or the sensation there, are exceedingly depressing and often lead a patient to a heart specialist and sometimes to an abdominal surgeon; this man had submitted to an appendectomy, but said that the operators found the appendix perfectly normal.

Dr. George Hall: I have observed cases of depression in which the patient was depressed for only a few days and then returned to normal for a short period, only to become depressed again. I had a patient in my office today who has been in a state of depression for several weeks, but during that period she would be depressed for one or two days at a time and then would experience two or three days of an apparently normal state. She would awaken with a feeling of nausea and lack of appetite, associated with extreme depression, which would last until late in the afternoon. Her mental condition would brighten up sufficiently so that she would be able to attend social functions in the evening, only to become depressed again on the following morning. She has run such a cycle for several weeks. The important symptoms Dr. Paskind has emphasized are: the feeling of nausea and the sudden depression and equally quick reversion to a period of well-being, each period lasting for only a few days.

Dr. A. B. Yudelson: I wish to know what Dr. Patrick and Dr. Paskind do for these patients?

Dr. John Favill: It may not be out of place to mention the other side of the picture. Recently, Dr. Alfred Solomon and I had a patient who since the onset of puberty has had two or three attacks a year of definitely manic nature, lasting only three or four days. This has puzzled every one in the family and help has not been gained through any medical aid that has been sought. The patient has a complete alteration of personality during these attacks, is foul mouthed and combative, and the attacks disappear as suddenly as they begin.

Dr. Paskind: As to the prognosis, I do not think there is any way of separating the short from the long attacks; the symptoms are identical. A few days' observation will reveal whether the attack is short or protracted, but the differentiation between a short and a long attack is not nearly so important as the differentiation between a short attack and a somatic disease or psychoneurosis. Here lies the source of not infrequent serious error. The treatment in the brief attacks is the same as that in the long ones. I do not doubt that many patients who have long manic attacks have brief ones also. They have been described by Gregory, and usually manifest themselves in excesses, principally inebriety.

STAINING OF TISSUES OF THE CENTRAL NERVOUS SYSTEM WITH SILVER

THE INFLUENCE OF THE STRENGTH OF THE REDUCING AGENT *

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In studies of the so-called "silver staining methods," emphasis has usually been laid on the rôle of the fixative and on the nature of the silver solution, while relatively little attention has been paid to the action of the reducing agent. Perhaps the first investigator to make use of wide variations in this step was del Rio-Hortega. In the gradual development of his method of staining microglia, he parted from the traditional reduction with 10 or 20 per cent commercial formaldehyde solution and found that selective staining of the cells could be secured by immersing the silver-impregnated sections, without washing, directly into a formaldehyde solution of only 1 per cent (1 cc. of concentrated liquor formaldehydi—approximately 37 per cent—in 100 cc. of distilled water). He also made certain interesting observations on reduction after a variety of intervening washes with, for example, alcohol, pyridine and other liquids. These observations, which I was privileged to share with him in his laboratory during the spring of 1926, have not yet been published.

OBSERVATIONS

My purpose in this article is to discuss the effects of varying the strength of the formaldehyde alone. These effects are most clearly seen when the sections are taken directly from the ammoniacal silver bath into the formaldehyde solution, without any intervening wash—as in the method of staining microglia. This procedure will therefore be discussed first.

The concentration of the formaldehyde solution has a primary effect on the speed with which the reduction occurs. On theoretical grounds this must be true; and, when tested either on solutions alone or on frozen sections, it is readily demonstrated that a weak solution reduces the silver slowly, while a highly concentrated formaldehyde solution of the same p_H reduces the silver rapidly. From this simple fact, many secondary results ensue.

1. Sections that are stained in any of the usual ammoniacal silver solutions will emerge from the silver bath with a color that varies from pale yellow to deep

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* From the laboratories of the Rockefeller Institute for Medical Research.

tobacco brown, depending on the duration, concentration and temperature of the stain. If such sections are then placed directly, without any washing, into an undiluted, fresh, approximately neutral solution of liquor formaldehydi (about 37 per cent formaldehyde), an almost instantaneous reduction occurs. This rapid reduction is characterized by three results: 1. The color of the sections after reduction is the same as before the reduction—at most becoming a trifle darker. 2. Under the microscope no precipitate is visible either on the sections or in the solution; this is true whether or not the sections are stirred during the reduction. 3. The tissues of the sections are homogeneously stained, so that little structural detail can be made out. The silver appears to have been taken up in an almost undifferentiated way, forming with the tissues a colloidal combination, the metallic units of which are smaller than the limits of microscopic visibility. In the flat field of yellow-brown, the cellular structures can sometimes be seen dimly as pale outlines or occasionally as slightly denser, browner bodies; but neither nerve cells nor fibers nor neuroglia are differentially stained.

2. If, on the other hand, some of the same sections are carried directly from the silver bath into dilute formaldehyde solutions (as, for example, a 1 per cent solution of liquor formaldehydi), a different picture results. In the first place, unless the section is vigorously stirred from the very instant that it enters the formaldehyde, an abundant gray-black precipitate will gradually form in the reducer, welling in clouds from the section. (With vigorous stirring, less precipitate will form in the formaldehyde.) During this process, the section itself slowly changes color—taking on a gray-black tone—the final intensity of which depends on the depth of the initial staining in the silver bath and on the dilution of the formaldehyde. There is a certain concentration of the formaldehyde at which this gray-black color reaches its maximum, while at dilutions stronger than this the sections will be yellow-brown, and at weaker dilutions they will be paler gray.

Under the microscope, these sections are seen to carry a gray, dustlike coating of finely divided silver against an almost colorless background. To some extent, this silver dust may be diffusely scattered over the surface of the sections. (This is particularly likely to occur if too weak a reducing bath has been used, or if the stirring has been inadequate during the reduction.) To a great extent, however, the fine granules of reduced metallic silver are seen to outline the cellular elements—nuclei, cytoplasm and processes.

COMMENT AND CONCLUSIONS

From these observations, it is legitimate to make several deductions:

1. The yellow-brown "argyrol-like" color of sections, after reduction in concentrated formaldehyde, is due to a true colloidal combination of the tissue and the silver, with the silver dispersed into units smaller than the limits of microscopic vision.

2. The gray-black color of sections after reduction in very dilute formaldehyde solutions is due (a) to the fact that during the slow reduction in weak formaldehyde the silver has had time to diffuse partially out of its loose union with the tissue elements, leaving a more or less colorless background; and (b) to the fact that with slow reduction the silver has had time to aggregate into particles that are of sufficient size to be visible under the microscope. Neither of these processes can occur under the more rapid action of the concentrated formaldehyde.

3. During the diffusion of silver out of the tissues which occurs in a weak formaldehyde reducing bath, the relatively loose and permeable structures of the background lose silver more readily than do the denser cellular elements. Reduction tends, therefore, to take place within the cells and on their processes and at their surfaces. (This interpretation is in harmony with the theories and experiments of von Möllendorf on the influence of diffusibility of dyes and density of tissue elements on the processes of differential staining by ordinary histologic dyes. It would be rash, however, to deny any influence to chemically selective forces in the differential staining of neurons and neuroglia with silver.)

4. Whether the reduced silver is visible as discrete granules, or is too finely divided to be seen as such, will depend on the density of the particular cells observed, the thickness of the section and the concentration of the reducer. This point makes it necessary to be extremely cautious in interpreting intracellular silver granules as "gliosomes," and granules or spicules of silver along the course of processes as true protoplasmic structures.

5. It is far harder to avoid the formation of misleading artefacts and of superficial encrustations and precipitates with the use of dilute reducing agents than with the use of stronger ones.[1] On the other hand, it is easier, with the dilute reduction agent, to pick out the parenchymatous elements for intensive staining. For any group of sections, therefore, it is possible to find an optimal reducing strength by trying out a series of formaldehyde solutions of different concentrations.

This optimal reducing strength is usually that at which the color-change of the sections from yellow-brown to gray-black is well discernible but not complete. In sections thus treated there is only a partial washing out of the background; and although there is some tendency for the silver to concentrate at the cell surfaces, there should be little aggregation of diffusing silver into microscopically visible particles of metallic silver dust. Occasionally, however, for special purposes (as for staining of microglia) a much weaker reduction than this will give the best results. In fact, the staining of microglia depends largely on this superficial deposit of metallic silver. With different sets of sections and different solutions, the actual optimal strength has been found to vary from a liquor formaldehydi concentration of 100 per cent (37 per cent formaldehyde) to one of 0.25 per cent (0.25 cc. of concentrated liquor formaldehydi in 100 cc. of water).

The optimal reduction must be determined by a few tests for each set of sections. One reason for this is that a number of variables other

1. Under appropriate conditions, cell-like and fiber-like formations can be approximated in coagulated egg-white by these methods.

than the concentration of the formaldehyde enter into the reaction, variables which are difficult to control; for example, the density and exact thickness of the sections, the p_H of the formaldehyde, the possible buffer content of the formaldehyde, the concentration and p_H of the silver solution, the excess ammonia of the silver bath, the heat and duration of the silver bath and so forth.

The same principles apply when the sections are washed between the silver bath and the reduction. In this case, however, much diffusion of silver out of the sections will occur in the washing, and the main effect that differences in concentration of formaldehyde exert is on the state of aggregation of the reduced silver. Here, too, however, optimal concentrations can be found which produce final stains that are superior to those produced by stronger or weaker reductions.

Abstracts from Current Literature

THE GENESIS OF GLIOMAS OF THE BRAIN AND OF THE LATE APOPLEXIES. RUDOLF BENEKE, J. f. Psychol. u. Neurol. **37**:22, 1928.

According to Beneke, by studying glial material the genesis of blastomatous formation can be better understood than by studying any other tissue. The development of carcinoma in connection with chronic mechanical irritation has been recognized for years, but the effect of chemical influences in the production of carcinoma has been accepted only during the last decade. The peculiar reactions of certain blastomas to some definite physicochemical stimuli is gradually assuming more and more significance in the problem of the origin of tumors. As far as glial tissue is concerned, the traumatic cases seem to offer most convincing evidence in showing how a hitherto normal tissue may undergo blastomatous proliferation, i. e., how it may become cataplastic.

Fundamentally, the combined effect of mechanical and chemical stimuli is as determining for the life and functions of glial tissue as for other tissues. The author attributes to glial tissue not only a mechanical supportive function but also the special ability to form and maintain the myelin sheaths of the nerve fibers; in this respect its function bears the same relation as myelin sheath cells do to peripheral nerves. The production of glial fibers is accordingly a response or reaction to mechanical stimuli within the brain. These mechanical stimuli have recently been shown by the author to be propelled in the form of irregular waves by the fluid pressure within the dural sac; they are brought about by external concussion (head movements, jarring during walking, chewing) on the one hand, and by internal concussion (arterial pulse) on the other. Ordinarily, the shocks induced by these concussions exert little ill effect on the glia within the normally dense structure of the central nervous system, because most of the force is spent on the nerve sheaths themselves. But with the advent of any condition which causes destruction of the nerve fibers there results a glial sclerosis sufficient to replace the softening induced by the necrosis of the fibers, although the glia cells at the margins of the necrotic area either remain in the same condition, or even proliferate (glial cicatrix). Beneke believes that this mechanism of formation of glial and ependymal scleroses is to a great extent influenced by the intensity and direction of the shocks of the fluid contents of the dural sac. It is also generally known that cicatricial sclerosis (the so-called apoplectic scar formation with more or less marked cystic degeneration) follows "softening." In this connection it must also be borne in mind that the glia cells are also subjected to irritative influences as soon as the nerve structure formed by it becomes necrosed. Marchand has shown that the so-called "fat granule cells" represent, to a great extent at least, glia cells that have been oversaturated with fat; in this respect the glia cells of the central organs resemble the myelin sheath cells of the peripheral nerves. Under such conditions, then, the glia cells take on an overabundant supply of lipoid. From a nutritional point of view, embryonal glia cells contain an enormous amount of that substance which is vital for cellular life, namely, cholesterin fat. This cellular overnutrition is not specially characteristic of blastomatous formation for it may also be seen in such conditions as fatty atheromatous aorta, kidney overloaded with lipoids, cholesterin tumor of the tunica vaginalis and other similar conditions without ever leading to the formation of a blastoma. On the other hand, Beneke had shown previously that growing tumor cells need large amounts of lipoid and that the greater the amount of lipoid locally or in the blood, the more rapid the growth of the blastoma. He believes, therefore, that the local accumulation of lipoids that have been liberated by the destruction (Waller) of myelin sheaths, such as occurs in injured nerve fibers within the white substance of the brain, favors proliferation of the adjacent living glia cells. This may suggest the possibility that the glia cells are possessed

of a special tendency to accumulate and elaborate lipoid substances biologically. It is this property, together with the chemical irritation as a result of an accumulation of an excessive amount of broken down products, which is frequently associated with the irritation of mechanical stimuli (shocks transmitted through the fluid within the dural sac) — conditions commonly encountered in cerebral softening — which seem to the author to be significant factors in the formation and growth of blastomas. It would seem then that the three favorable factors for the growth of blastoma (glioma) are: (1) space for expansion; (2) mechanical irritation, and (3) chemical changes.

This conception, according to Beneke, would have been accepted long ago, were it not for the well known fact that it is only in relatively rare cases that gliomas follow cerebral softenings. The author then goes on to cite two cases which are typical of late apoplexy following primary cerebral softening; they represent a large series of cases in which apoplectic scars follow a red or a white softening full of proliferating glia cells and sclerotic fibers, but with no progressive gliomatous proliferation. It would seem, then, that in cases of glioma there must exist some additional factor which changes a simple reactive proliferation into a blastoma. In the present state of knowledge the nature of this factor, whether local or constitutional, cannot be determined. In the light of the results obtained in experimental tar cancer, Beneke is more inclined to favor the existence of certain chemical products of decomposition rather than of constitutional differences which influence the reacting capacity of glia cells. One would then have to assume that cerebral softenings are not resorbed with equal rapidity in all cases, so that the time element for the carrying off of the products of decomposition would depend to a great extent on the site of the softening (e. g., near the pia), and the duration of the irritation would accordingly be a prominent factor in the entire process. Speculative as this theory may appear, nevertheless one cannot fail to recognize the fact that clinically as well as anatomically in almost all of these cases there is order to the sequence of events in the development of glioma following trauma, both as to time as well as to the location of the tumor. A careful history almost never fails to elicit this chronologic order of events.

As far as the development of a white softening following the various traumas is concerned, it is well to bear in mind that it has been generally accepted that in certain organs the closure of an artery, even as short in duration as in arterial spasm, will be followed by a local asphyxia which may lead to more or less necrosis of sensitive cells. The brain is no exception to this rule. The occurrence of ischemic cerebral necrosis during birth injuries is, according to Beneke, a good example of such causal relationship. This necrosis, according to him, can be explained only on the hypothesis of vascular spasm. He has observed several cases which support anatomically the hypothesis that various kinds of trauma give rise to local cerebral necroses following ischemic softening. He believes, therefore, that in these traumatic cases the degree of damage to the cerebral white substance will depend on the duration of the ischemia. The greatest damage conceivable would be a total necrosis with complete destruction of the corresponding glia cells, although even in such a severe case the margin of the lesion may be expected to show glia cells which, though damaged, have not been totally destroyed. As a matter of fact, in such areas one actually finds only broken down myelin sheaths without actual necrosis of the glia cells. The latter very likely recuperate from the injury, and, having done so, they have a great deal to do with the process of resorption and reparation of the degenerated myelin sheaths (marginal proliferation of an ischemic focus). One could also conceive that in some cases a certain part of the brain is so little affected by the ischemia that there is proportionately very little glial involvement so that the degenerated nerve fiber system would be permeated by glia cells that are still living. To what extent such a condition would favor proliferation becomes evident when one takes into consideration the foregoing observations as to the significance of lipoid nutrition of the glia cells and the free space gained through nerve resorption.

Unfortunately, the very nature of the subject does not permit the author to produce histologic evidence as to the truth of his hypothesis. The preliminary stages of proliferating glioma disappear so rapidly that they do not lend themselves to histologic demonstration.

KESCHNER, New York.

THE INFLUENCE OF THE ROENTGEN RAYS ON TUMORS OF THE BRAIN. O. MARBURG, Arb. a. d. neurol. Inst. a. d. Wien. Univ. **30**:171 (Aug.) 1928.

In spite of many reports of improvement and recovery from tumors of the brain following roentgen therapy, the actual process involved is not entirely clear. For many years Marburg has believed that the influence of the rays was a mechanical one affecting intracranial pressure. Of all the tumors of the brain, glioma is most amenable to roentgen therapy because here one is dealing with an extremely cellular, rapidly proliferating tissue, and the tumors usually are solitary.

Marburg describes three cases in which gliomas were treated with the roentgen rays and later removed at autopsy. Each tumor had been treated in several fields, usually six or eight, with three-quarters the maximum skin unit dose.

The first case was a cystic glioma of the brain. Examination of a typical area showed it to be composed of characteristic cytoplasmic glia cells which were connected with each other by a dense fibrous network. In some of the cells the cytoplasm was somewhat larger than in others, and it contained fine acidophil granules. Occasionally, the contour of the cell was lost and the granules lay free. Other cells were rounded off and resembled gitter cells. In another area there was a dense whorl-like glial network; the cells were poor in protoplasm and the nuclei underdeveloped. It looked almost as if there were naked nuclei although one could distinguish a slight protoplasmic membrane. These dense glia lay, for the most part, around small cavities which were filled with a homogenous mass. The whorls of glia appeared to be a reaction to the cavitation. Around the vessels one could occasionally recognize endothelial characteristics in the cells.

Therefore, this was a type of glioma with cyst formation and was different in no way from the usual picture of such a tumor. With the exception of some cells containing acidophilic granules and finally dying, there was nothing attributable to the influence of the roentgen rays. There were two types of these cells, one kind resembling gitter cells, and the other partaking of the character of cytoplasmic glial cells.

The patient in case *2* received more treatment than the one in case 1. The condition was a glioma of the temporal lobe. In typical areas this tumor differed in structure from that in the first in that it was more cellular. However, the cells were also glia cells. Their nuclei showed no signs of degeneration, and there was both mitotic and amitotic cell division. However, there was one striking observation. In areas in which the tumor was unusually cellular there were cells with a considerably enlarged cytoplasm. Near the periphery of the tumor these cells greatly increased in number so that in places they occupied the entire area. They were either uninuclear or polynuclear and resembled mast cells. Some of these cells were disintegrating, but most of them were actively engaged in fibrillogenesis. In this area one could see the small tumor cells being replaced by dense glial network. In places this network was less dense, and beginning necrosis was apparent.

A second unusual observation in this case was seen around the blood vessels in the form of endothelial-like cells which appeared in groups and heaps and at the same time showed signs of degeneration. They were undoubtedly related to the vessels. The tumor was unusually vascular, and the vessels were hyperemic with a tendency toward hemorrhage.

The peculiarity of this second case was that after the degeneration of tumor cells there were found cells which are observed also in encephalomalacia. Besides this there were islets of glial fibers with central necrosis and sclerosis. These were devoid of cells and were surrounded by large cytoplasmic glial cells.

Case 3 was a glioma of the frontal lobe of over six years' duration. This patient was irradiated and then operated on. At operation there was a hemorrhage that

resulted in a hemiplegia, which improved under further irradiation systematically carried out for three years, when the patient died apparently from acute cerebral edema.

As in the first two cases, the tumor was composed primarily of glia cells. All forms, from those with only a small amount to those with an abundance of cytoplasm, were visible. Two types of cells were present — typical degenerating cells, and cells showing fibrillogenesis. The tumor was a cystic glioma and showed an unrestricted growth where the cells assumed the most varied forms. Only toward the periphery where the tissues were looser could one distinguish any cytoplasmic cells. As in the other two cases, these showed active fibrillogenesis with massive cells and formation of glial plaques. At times necrotic areas could be seen, but on the whole the structure resembled a glial scar. In some places the tumor appeared as if it were composed only of large glia cells. At the same time, the section showed an uncommonly cellular form of tumor which did not have the usual appearance of a glioma.

When one views these three cases, it is obvious that neither a single irradiation nor an irradiation extending over a period of years can inhibit the growth of a glioma. The question now arises as to what phenomena can be interpreted as signs of the effect of the roentgen rays. Even nonirradiated gliomas contain dysplastic glia cells. Marburg believes, however, that in these cases there is something more than simple cellular degeneration or exhaustion (Abbau), for in the cases in which irradiation was done, only once were these cells isolated and they had more the character of real degenerating cells. However, in the second case there were large cytoplasmic cells surrounded by a dense glial network simulating a scar. In this region there were not any tumor cells. This type of reaction was still more pronounced in the third case in which irradiation had been practiced over a period of several years. Simultaneously there was necrosis of the fibers with cavitation of the tissues. Still another fact is important. This is that these glial scars were most prominent at the periphery of the tumor. The question now arises as to whether the tumor by its enlargement could not have produced softening in the surrounding tissues with the resultant formation of cytoplasmic glia cells and glial scars. Marburg does not completely refute this theory although he found areas in the interior of the tumor which were identical with the periphery.

The blood vessels were no different from those found in ordinary gliomas.

The question now arises as to whether the cyst formation could be regarded as a result of the irradiation. In case 3 the cysts apparently were the result of necrosis following some external influence. While Marburg believes himself to be alone in the view, he thinks that this necrosis may be attributed to the influence of the roentgen rays.

Cases reported by Roussy, Labarde and Levy correspond in many respects to those described by the author, and the general opinion is that the changes in the proliferating element are the result of the roentgen rays.

In conclusion, Marburg states that there is no definite evidence that the roentgen rays exert a particularly destructive effect on gliomas and that the reactive changes and sclerosis should be regarded as reparative. He believes that the principal effect of the rays is not on the tumor itself but influences primarily those factors favoring increased intracranial tension. This effect probably is on the plexus cells.

KAMMAN, St. Paul.

THYMOGENOUS CAPILLARY ATONY AS A CAUSE OF CEREBRAL DISTURBANCES. CARL WICHURA, J. f. Psychol. u. Neurol. **37**:401, 1928.

A well developed, obese farmer, aged 55, with unusually large extremities, after an entire winter's inactivity, became more obese and began to have periods during which he suffered from headaches, depression, anxiety, insomnia, muscular contractions, generalized "sticking" pains and paralyses involving one of his upper extremeties and half the tongue. These symptoms came on periodically and would last for weeks at a time. At the beginning of the spring he became active again and after having been subjected to a "reducing" diet began to lose weight and

regained his usual physical and mental health. As the symptoms began to regress he was noted to have developed a marked atony of the capillaries at the margins of the nails. With complete recovery this phenomenon had also entirely disappeared. The only organic observation in the case was a marked polycythemia.

According to Wichura, this case seems to confirm Westphal's theory of the angiospastic origin of apoplexies and may serve as a contribution to the knowledge of the apoplexies sine materia.

The cerebral manifestations presented by the patient could justify the assumption that one was dealing here with a certain amount of increased intracranial pressure, whereas the mental symptoms were extremely suggestive of a transitory Korsakoff syndrome. Fever, uremia, toxic or expanding intracranial lesions could definitely be excluded as causative factors. The rapid disappearance of the nervous symptoms, as well as the changes in the blood picture following the general improvement, would also speak against the polycythemia as an etiologic agent. A study of the blood picture was also suggestive that one was dealing with a symptomatic polycythemia which, according to Nägeli, must be regarded as a secondary manifestation of disturbances of the endocrines. There was nothing in the history to point to the possibility of a transitory polycythemia following poisoning by phosphorus, carbon monoxide or trichinosis, nor was there any evidence of tuberculosis of the spleen.

From the point of view of constitution it was noteworthy that the patient's family consisted of strong, long-lived, tall and obese persons, although there was some familial tendency to tuberculosis. Except for the unusually large extremities his physical development was normal. There was no evidence of defective development or hypofunction of the genitalia or of the thyroid. The disproportion in the size of the body and limbs with the obesity and vascular atony could not be attributed to any hypophyseal disturbance, because closer study revealed that the skull was normal; there were no abnormal distribution of the body fat or disturbances in the urinary excretion. Furthermore, although the patient's mood could be interpreted as a sequel to cerebral circulatory disturbances as well as to hypophyseal dysfunction, the latter would have to be in the nature of a hyperfunction of the anterior lobe to account for the large growth, and of a hypofunction of the middle and posterior lobes to account for the obesity. To explain the entire clinical picture one would also have to find some reason why the second tonus mobilizer of the blood vessels — the suprarenals — temporarily failed to perform their function. All in all, any attempt to attribute all the clinical phenomena in this case to an actual disturbance of the hypophysis would encounter too many objections.

Wichura believes that an involvement of the thymus would best explain the entire clinical picture. According to Biedl, hypofunction of this gland is characterized by unusual body height with very long extremities and a relatively short trunk, a well developed panniculus adiposus and a tendency to tuberculosis — all of which features were present in this case. The thymic involvement may also have been a factor in the production of the vagotonia and, as the opposite to the two tonus maintaining agents (posterior lobe of the hypophysis and suprarenals), to the vascular atony. The fact that lymphatic glands were not demonstrable clinically in the patient may have been due to their disappearance before the patient reached his present age, or to an absence of the lymphatic component of his thymic constitution. Of interest in this connection also are the high lymphocytic count (34 per cent) and the high thymus figures obtained by Abderhalden's interferometric examination, even though no enlargement of the thymus could be made out clinically. Retrogression of gonadal activity, lack of muscular effort, obesity due to overeating and the time of the year during which the patient became ill (winter) were probably additional factors in the thymus dysfunction. The latter was probably a transitory phenomenon, because as soon as the patient's weight was reduced and with the onset of warm weather he was subjected to cooling tonic carbonated baths during which increased muscular activity was encouraged, his symptoms improved rapidly.

The capillary atony, as observed microscopically, exceeded in intensity that of any other case observed by the author in thousands of patients subjected to such

examination during the past ten years. One glance under the microscope was at once suggestive that it was the capillary atony that was at the basis of the patient's various circulatory disturbances. Wichura believes that all cerebral vessels were affected, and the more pronounced paretic phenomena he attributes to quantitative differences in the circulatory disturbances of the cerebral cortex. What effect the chemicotoxic changes of the blood and body juices had on the cerebral manifestations must, in the present state of knowledge, still remain an open question.

KESCHNER, New York.

PSYCHOLOGIC DETERMINANTS OF CASUALTIES DURING WAR. A. KLUGE, Arch. f. Psychiat. **84**:739 (Sept.) 1928.

It is established that the occurrence and frequency of accidents are related to personality types; that certain types of persons can be shown statistically to be more prone to meet accidents than others. This factor was taken as the basis of a study of psychologic personality determinants of casualties during the World War. The author found that the predisposition to suffer casualties was related not only to certain individual personality types, but also to certain groups in the army. These groups can be classified as follows:

1. According to the social status of the person: (*a*) Leaders. In the upper classes the danger of injury is increased by the fact that these persons have to serve as an example to the men they lead; therefore, they expose themselves to danger without showing signs of fear. This, however, is compensated for largely by the fact that as a rule these leaders are of higher intelligence than the others and can act more quickly when facing danger; also, the possibility of psychopathies and psychoses is less as they have been weeded out during the process of advancement. In general, therefore, there is less danger here than in other stations. The compensating factors mentioned in the cases of higher leaders lose their value in men of the lower classes and in noncommissioned officers; the desire to advance brings with it also a tendency for more reckless exposure, and here there is consequently a large percentage of casualties.

(*b*) The rank and file. Here a predisposing factor is lower intelligence and inability to act on personal initiative; these, however, are compensated for by the fact that the man is not called on to exhibit any special courage.

(*c*) The difference between actively engaged soldiers and those behind the lines is also evident. Whereas the man on the firing line is in constant danger, those behind the line are not so alert or so keen to the presence of danger as the former and therefore are not so careful.

2. The branch of the army: (*a*) The most reckless, as well as most careless, are first the flyers and then the marines. The exciting and changeable life of these two types of service attracts many psychopaths and adventurers, and so one finds among them a high percentage of casualties.

(*b*) The cavalry is an almost extinct branch of the army, and was not of so much importance in the World War as in previous wars. At the beginning of the war there was a certain amount of tradition and romanticism about the cavalry, with a concomitant tendency for reckless charges and a consequently larger percentage of casualties. This, however, decreased toward the end of the war, and at its close the behavior was much like that of the infantry.

(*c*) The artillery. The men in this service are of higher intelligence, and they are better looked after than in any of the other branches. They are cooler and more deliberate than the others, and one finds here a lesser possibility of casualties.

(*d*) In the infantry, the greatest danger lies in the fact that individuals do not mean much; the mass is dependent on single accidental occurrences. It is also here that the danger is most pronounced because of an inability of the infantryman to act by himself and a tendency to behave as the mob does. Among other factors of importance, the type of warfare influences the danger of casualties. The danger is increased during charges; it is enhanced by disease, vermin and uncleanli-

ness. It is also influenced by the predisposition of certain persons to the development of mental disease, by the attitude that the men show toward being taken prisoners, and so on.

The author reaches the following conclusions: 1. The danger of casualties in war is increased by certain psychologic influences; with a knowledge of these influences it is possible not only to predict such dangers but to decrease them.

2. It is important for the military surgeon to realize these factors and if possible to remove them.

3. It is advisable that officers in the army be instructed concerning these dangers.

MALAMUD, Foxborough, Mass.

ON THE LATENCY OF NEGATIVE AFTER-IMAGES FOLLOWING STIMULATION OF DIFFERENT AREAS OF THE RETINA. R. S. CREED and RAGNAR GRANIT, J. Physiol. **66**:281 (Nov.) 1928.

The authors' investigation is concerned with the latent period preceding the ordinary persistent after-image which is readily seen when, after fixation of any well defined object, the gaze is transferred to a point on a uniform background. The experiments were carried out in a photographic dark room, lighted by a single opal glass electric lamp of about 80 candle power at one end of the room. Before any observations were made, twenty or thirty minutes were allowed to elapse so that the observer's eyes might become adapted to the illumination. The condition of the eyes in this illumination may be described as one of moderate light adaptation. Against the evenly black wall at the opposite end of the room from the lamp, white circular disks, provided with a fixation point, were observed binocularly. The disks were of paper coated with a mixture of zinc oxide and barium sulphate, and their illumination worked out at just over 5 meter-candles. The observer's eyes were 1.50 meters from the disk, and the time during which the disk was fixated was fifteen seconds. Before each fixation period, the observer shut his eyes for thirty seconds in order to get rid of disturbing effects caused by after-images of objects in the room.

Using small disks of constant size at various distances from the fixation point, they found that the latent period is longest in the center of the field of vision, shortens rapidly to a point about 2 degrees from the fixation point, then increases to a new maximum between 2 and 3 degrees from the fixation point, and thereafter again shortens. The second maximum or "hump" in the curve is interpreted as being due to the replacement of the cones by the rods as the dominant receptor organs in and beyond the area. Changes in the appearance of the after-image lend support to this idea.

The problem next investigated was the relation between the latent period of after-image and the size of the disk, other conditions being kept constant. Disks up to 22 cm. were used. It was found that a precisely similar curve was obtained by plotting the visual angle subtended by the radius of the disk as abscissa and the latent period as ordinate. The latency of the after-image of the disk as a whole is that corresponding to the region of the retina on which the image of its edge falls. Large disks gave after-images with more phases and a longer total duration than did small, but their contours were less sharp. Phases after the first were always less black and less well defined. Some part of the edge was always the first portion to appear and the last to disappear. The latent periods of after-images of disks with their centers in the periphery of the field of vision are, within certain wide limits, independent of their size.

Creed and Granit believe that two important principles are illustrated. One is the importance of form, exemplified by (*a*) the filling in of areas and contours in a blind region of the field of vision, and (*b*) the observation that the length of the latent period preceding the development of a negative after-image is dependent on the region of the retina on which the edge of the primary object falls, and is independent of the position of other parts of the image. The second principle is the interaction which occurs between the various receptors stimulated

by a primary image. In their experiments this manifested itself as inhibition of the cone mechanism by the rod mechanism. No information is available as to the anatomic site of the process, but in view of the recently published work of Adrian and Matthews on the interaction of neurons in the eye of the conger eel it may well take place in the retina.

ALPERS, Philadelphia.

A CLINICOPATHOLOGIC STUDY OF ALZHEIMER'S DISEASE. E. HERZ and E. FUENFGELD, Arch. f. Psychiat. **84**:633 (Aug.) 1928.

Since the first description of this disease by Alzheimer and Kraepelin, numerous authors have studied cases of this type but have added little to the clinical and anatomic picture originally described. The typical clinical picture of psychomotor unrest, speech disturbance and rapid deterioration, as well as the concomitant histologic changes, have always been regarded as capable of showing little variation; consequently, no other study was undertaken. In this article the authors report a study of the clinical symptoms with the purpose of gaining a better insight into their meaning and their possible relation to the histologic processes. Three cases are reported. In all, the clinical picture showed all of the typical symptoms of Alzheimer's disease: increased activity, rapidly progressing dementia, some focal signs and disturbance of memory. It seems that in the motor phenomena one is dealing primarily with two definite components: (1) the peculiar motor excitement (hyperkinesis); (2) a tendency to iterative repetition. The author distinguishes two types of so-called motor unrest: (1) psychosensory, and (2) psychomotor hyperkinesis. The former is best shown in the so-called occupation unrest in which the patient attempts to perform definite acts. The latter is much simpler, does not seem to depend on outside stimuli and in its simplest form dwindles down to movements of isolated parts. Observation of the development of Alzheimer's disease shows that patients begin with a more complicated psychosensory unrest which in time breaks down into movements of isolated parts of the body, but which probably represent parts of an earlier, more complicated pattern. Because of this meaning as a part of a definite and complicated reaction, they may be preserved as representatives of such activity and later in the disease be automatically repeated in an iterative fashion. A similar interpretation is advanced for the speech disturbance. Early in the disease there may be paranoid or other trends which, even if pathologic, still can be followed. Gradually these complex utterings break down into more or less isolated words, and as a result there develops the so-called logoclonia.

Microscopic studies from a topistic point of view indicate that it is possible to find a relationship between the clinical picture and the degree of involvement of different systems in the brain. Kleist has advanced a view that diseases of definite centers in the brain are related to certain types of motor unrest. The psychosensory hyperkinesias are due chiefly to disease of the thalamus, whereas psychomotor disturbances (in the sense mentioned) are due to disease of the large cells of the caudate nucleus. It is possible, therefore, that in Alzheimer's disease, in which psychosensory disturbances are found at the beginning of the disease but largely disappear and give way to psychomotor symptoms, one is dealing with a disease of the thalamus at the beginning and a gradually increasing disease of the caudate toward the end. Both of these centers were definitely affected in the cases studied; so far, however, no basis has been found for a statement as to which was involved first. The vegetative disturbances during the course of this disease — loss of weight, sleep disturbances, etc. — are considered as related to a marked predominance of microscopic changes about the substantia grisea centralis. A discussion of the etiologic possibilities leads to the conclusion that nothing definite can be said. It does seem as if the Alzheimer fibrillary cell changes could be produced without the senile plaques, and vice versa; the causes of these two changes may be different. The authors believe that there is not enough evidence to justify consideration of the possibility of an endocrine origin for the disease.

MALAMUD, Foxborough, Mass.

CONSTITUTIONAL PSYCHOLOGICAL FACTORS IN "FUNCTIONAL PSYCHOSES": II. DEMENTIA PRAECOX. HELGE LUNDHOLM, J. Nerv. & Ment. Dis. **68**:456 (Nov.) 1928.

The author distinguishes two tendencies in the normal person, known as the altrocentric and the egocentric. The former includes such traits as extraspection and extra-activity, and altruism or sympathy. The latter comprises introspection, seclusiveness and egotism. The A tendency furthers the race, and the E tendency furthers the individual. In the A tendency, both self-assertive and submissive impulses are favored, whereas the E tendency excludes the submissive entirely.

In dementia praecox, there is a strong egotistic tendency and a consequent absence of altruism. This egotism is seen in the positive seeking for self-satisfaction and in the negative avoidance of self-injury. The schizophrenic person will not suffer the burden of the normal social man. Submission is a social relation painful to his egotism, yet this submission is the essence of social adaptability.

In analyzing cases of dementia praecox, two types are to be found: (1) the egotistic, introspective, seclusive type, preoccupied with his own meditations; (2) the egotistic, introspective, extra-active type. The silly, hebephrenic praecox patient seems to conform to this latter type. The paranoid schizophrenic person with seclusiveness who, nevertheless, displays socially his delusions of persecution, conforms also to the latter group. In cases of schizophrenia, depressive reactions may manifest themselves in suicidal attempts; often also in the form of neurasthenia-like states probably resulting from the exhaustion of an emotional conflict. The presence of these features of manic depressive insanity in cases of dementia praecox is exemplified in the so-called mixed disorders. Patients with manic depressive insanity and dementia praecox show a strong constitutional E tendency.

The author considers that in cases of catatonic stupor there is much greater extraspective activity than there is in more demented persons; the catatonic patient follows the environmental happenings with keen interest and maintains an attitude of tenseness in the presence of others. He likens the inner world of the schizophrenic person to a sphere in which the outer layer represents reality and the inner parts fantasy fused with reality in decreasing proportions as one nears the center. In dementia praecox, the patient lives in a reality which has a fusion of imagination and objective environment. The author considers that the tendency to fuse reality and fantasy is the essence of the schizoid trait; the splitting in these cases produces a cessation and solution of the conflict. The distress of the neurasthenic person, on the other hand, is due to his inability to solve his conflict either in this or in any other way. However, the schizophrenic patient, before his splitting, usually shows this neurasthenic picture. His conflict, however, is usually due to his strong egotistic tendency, his inability to submit to others, his increasing sensitiveness and a tendency to exaggerate everything that goes against him, developing later into feelings of persecution. The fusion of objective reality with fantasy occurs through the disappearance of the ability to recognize the normal criterion of realness, namely, the resistance of the objective world. The schizophrenic patient seems to differ from the normal man in the fact that he does not apply this test to his experience, but accepts objects of both perceptual thinking and fantasy as true reality. The author sees no essential difference between the dissociation of the hysteric and that of the schizophrenic person; both serve to relieve mental pain, the dissociation in the former is simpler and there is better balance of the A and E tendencies than in schizophrenia. This enables the hysteric patient to make the submissions necessary for social adjustment.

HART, Greenwich, Conn.

RELATION OF OTOLARYNGOLOGIC DISEASE TO MENTAL DISEASE. G. B. M. FREE, Arch. Otolaryng. **8**:707 (Dec.) 1928.

The majority of investigators in the field of mental medicine are of the opinion that the mind can no longer be considered as a separate entity. The theory that mental diseases are the result of toxic disturbances from foci and other organs

of the brain is gaining widespread credence. With this in view the author, Assistant Superintendent of the Danville State Hospital, Danville, Pa., has examined the nose, throat and accessory sinuses of patients with mental diseases and has made the following observations: These mental patients do not express themselves in the same fashion as normal persons. "The expressions are not 'I have pain over my eyes or over my cheek' but 'someone is passing electrical currents through my skull or my face,' and he reacts to these by devising all sorts of mechanical treatments that he applies himself." Frequently, they attempt to treat themselves with foreign bodies such as chewing gum and wedges of wood which have been found in cases that appeared to have been sinus trouble. Relief of the sinus condition shows an amelioration of the mental state. A mild mental disorder may advance to a well defined psychoneurosis of the neurasthenic type.

"It would therefore be well to remember that the so-called neurasthenic patient with headache and pain in the neck and down the spine, who complains of inability to remember names and objects and who has become careless, indolent and wholly indifferent to everything except himself and his symptoms, may have an overlooked sinusitis, and that the securing of proper drainage and aeration of the sinuses may result in a prompt disappearance of the physical discomfort of the mental symptoms."

The author has noticed the frequency of erysipelas in hospitals for the mentally defective and says that it is found especially in persons with dementia praecox and in mental defectives and senile groups, in other words, among the unclean and untidy patients whose mental condition renders them incapable of caring for their personal hygiene. "Examination of these patients frequently reveals an infection of the mucous membrane of the nose and accessory sinuses."

A patient complaining of respiratory symptoms and inward goiter was relieved within forty-eight hours after tonsillectomy. The author rarely finds that tonsillar disease is the sole determining factor, but it aggravates the underlying condition.

"One of the important relations existing between infected tonsils, overgrowth of adenoid tissue and mental functioning is found in the case of children.

"It is surprising to see the large percentage of boys and girls considered mentally subnormal even to the degree of mental deficiency, unable to make any progress in school, dull, stupid and indifferent, who have passed through the extramural mental clinics of this hospital, and who after the removal of tonsils and adenoids, have become normal in every respect."

Conclusions.—(1) A definite and dependable relation exists between physical disease and abnormal mental functioning. (2) Thorough physical examinations should form a part of the investigation of every patient presenting mental symptoms. (3) In mental diseases, infections of the nose, throat and accessory sinuses frequently form a part of the symptom-complex; diagnosis and treatment in early cases may prevent the development of a psychosis, and in patients already hospitalized may contribute to comfort and sometimes to cure. HUNTER, Philadelphia.

A CASE OF ABSENCE OF THE CORPUS CALLOSUM. MAX DE CRINIS, J. f. Psychol. u. Neurol. **37**:443, 1928.

A primipara gave relatively rapid and easy birth to a monster with a huge meningocele, deformed hands and feet, harelip and cleft palate. The monster showed no gross evidences of motor or sensory disturbances. It lived three weeks. At necropsy the brain appeared as follows: the frontal poles were swollen and protruded into the meningocele; the left hemisphere was longer than the right, which was tougher in consistence and club-shaped; the left temporal lobe was also considerably larger than the right, but the right occipital lobe was broader than the left; marked microgyria and polygyria; total absence of the corpus callosum, the roof of the third ventricle consisting only of a delicate membrane. The lateral ventricles were unusually dilated and extended as far as the most anterior portion of the frontal poles. The meninges at the base showed early evidences of an inflammatory process; the optic tracts and nerves were markedly atrophic, and the chiasma was flattened. The pons was rudimentary and so

flattened out that it was barely raised over the basal part of the cerebellum. The cerebellopontile angle could hardly be made out, and the lower cranial nerves appeared in the form of fine atrophic strands. The cerebellum itself was also asymmetric, and together with the medulla oblongata was pushed by the right occipital lobe to the right. Anteriorly, the optic thalami could hardly be distinguished from the basal ganglia. The ependyma of the ventricles was rough and thickened.

Histologic examination revealed the following: A Nissl preparation of a section from the ventricular wall showed small subependymal collections of glia cells interspersed with polymorphonuclear giant cells. Numerous masses of pus cells were found within the walls of the ventricles as well as in the uppermost layers of the cortex; these collections of pus cells were surrounded by small encephalitic foci. Nerve fiber preparations showed in many areas that the nerve fibers were still nonmyelinated. This, according to the author, is of great significance because the sections were obtained from the region of the anterior central gyrus in which one would ordinarily expect early myelin projection tracts in an infant aged 3 weeks. In contrast to this, the mesial surfaces of the cerebral hemispheres showed relatively well developed bundles of myelinated nerve fibers. Four such bundles, sharply separated from each other, could be made out in a relatively small area in this region; in frontal sections made more caudad these bundles changed their direction and grouped themselves around two areas which appeared unusually pale and represented most probably accumulations of nuclei. These fibers were most likely rudimentary fibers of the internal capsule and the nuclear accumulations, the poorly developed basal ganglia. Unfortunately, the preparations did not permit an investigation of the fasciculus callosus longitudinalis; in cases of absent corpus callosum, this fasciculus is almost always poorly developed.

The author believes that the internal hydrocephalus in this case is not only a malformation but is also the cause of the developmental defects. According to him, internal hydrocephalus not only prevents the development of the corpus callosum, but also may cause its disappearance after it had already been completely developed.

KESCHNER, New York.

THE INFLUENCE OF CEREBRAL BLOOD-FLOW ON RESPIRATION: I. THE RESPIRATORY RESPONSES TO CHANGES IN CEREBRAL BLOOD-FLOW. CARL F. SCHMIDT, Am. J. Physiol. **84**:202 (Feb.) 1928.

This is one of three papers reporting work which was designed to test the hypothesis that the pulmonary ventilation is largely dependent on carbon dioxide pressure within the cells of the respiratory center. This obviously is dependent on several factors: (1) the concentration of the stimulant material in the arterial blood; (2) the rate at which the stimulant material is produced within the cells of the center, and (3) the rate of blood-flow through the center.

Dogs and cats were used as experimental animals, and three methods were applied: the measurement of arterial inflow to the brain, the measurement of venous outflow from the brain and perfusion of the brain. The author shows nineteen records from his experiments; only one protocol is given. The records illustrating the effect of "acute cerebral anemia" are especially interesting. He summarizes by saying:

"1. Methods are described for measuring cerebral blood-flow as arterial inflow and venous outflow, and for perfusing the brain with a measurable volume of blood, in spontaneously breathing cats and dogs.

"2. Evidence is presented that cerebral blood-flow varies with changes in systemic blood pressure and is not subject to direct vasomotor influences of sufficient strength to be detected by these methods. The cerebral vessels are shown to be under intrinsic chemical control, for they are dilated by CO_2 anoxemia, acid, heat and cerebral anemia, constricted by excess oxygen and alkali, cold, and probably by increased blood-flow.

"3. Respiration is depressed by an increase in cerebral blood-flow, stimulated by decrease, no matter how produced, though within certain limits only. Respira-

tory depression follows a marked increase in cerebral flow even during CO_2 or nitrogen inhalation. Similar results are obtained when the vagi are cut, and whether systemic blood pressure rises or falls, showing that reflexes from the heart and aorta are not the sole cause of the respiratory response; it is also shown that a rise in blood pressure may have little or no effect on respiration until cerebral blood-flow is allowed to increase. The respiratory responses are not due to changes in intra-cranial tension.

"4. Beyond a certain limit a reduction in cerebral blood-flow depresses breathing and an increase then stimulates respiration; to this condition the term Reversal is applied. It is shown to be due to an insufficient supply of oxygen to the brain, and can be produced by decreased blood oxygen content, by decreased cerebral blood-flow, or by stimulation (electrical, by drugs or by CO_2 inhalation) in the presence of reduced cerebral blood-flow. It is prevented or removed only by an improvement in cerebral oxygen supply, most effectively through an improvement in cerebral blood-flow.

"5. These results explain the paradoxical relations between blood pressure and respiration which led to these experiments, and are presented as additional support to the theory of Gesell."

THE INFLUENCE OF CEREBRAL BLOOD-FLOW ON RESPIRATION: II. THE GASEOUS METABOLISM OF THE BRAIN. CARL F. SCHMIDT, Am. J. Physiol. **84**:223 (Feb.) 1928.

This paper reports a comparison of the gas content of blood flowing into and out of the brain under different conditions of cerebral activity, the activity being measured primarily by the state of the respiration. Estimation of oxygen consumption and carbon dioxide production of the brain is the best criterion of metabolism, but the determinations are difficult because an isolated circulation must be prepared, arterial and venous blood must be collected without interfering with the circulation of the organ, and accurate estimation of the volume of blood-flow corresponding with the samples of blood must be made. The method employed was perfusion of the vertebral arteries. Epinephrine was added to the perfusing blood, and the pump was set at an output which maintained perfusion pressure above systemic. These procedures eliminated, as far as possible, errors arising from the escape of blood into extracranial vessels, and from the inflow from extracranial vessels. The technical methods show great skill, and the operative procedure was evidently difficult.

Changes in respiratory activity were brought about by electrical stimulation of the medulla, of the vagus, and by punctures of the medulla. The inhalation of carbon dioxide was also used, and the effects of such drugs as morphine, atropine, caffeine, ether and ephedrine were studied. The results are given in tabular form. A curve summarizing all the data shows in a striking way that cerebral metabolisms appear to vary with the activity of respiration. An interesting suggestion is propounded to the effect that since carbon dioxide, acid and oxygen lack all dilate vessels, there may be local variations in blood-flow, the more active parts of the brain having relatively greater blood-flow than the inactive portions. In these experiments, for example, a hypothetical picture of the tissues would probably show "a number of small scattered areas with a high metabolism rate, surrounded by a huge mass of cells with a very low metabolism. These would represent the parts concerned in maintenance of respiration, circulation, etc., on the one hand, and the narcotized 'higher centers' on the other. It is not only possible but highly probable that the distribution of blood among these parts should be subject to fluctuation from instant to instant: we have shown in the preceding paper that such vascular readjustments are possible."

THE INFLUENCE OF CEREBRAL BLOOD-FLOW ON RESPIRATION: III. THE INTERPLAY OF FACTORS CONCERNED IN THE REGULATION OF RESPIRATION. CARL F. SCHMIDT, Am. J. Physiol. **84**:242 (Feb.) 1928.

The last of these three papers is, as its title implies, a discussion of the various factors controlling respiration: (1) the influence of changes in cerebral vessels;

(2) the significance of the vasomotor response, and (3) the part played by changes in the metabolic activity of the center. Periodic respiration is also discussed. In summarizing, the major conclusion is stated as follows: "The chief function of the entire respiratory mechanism is the maintenance within the cells of the center of a constant concentration of stimulant material. The factors involved in this maintenance are those enumerated in our tentative hypothesis, namely: First, changes in the arterial blood, produced chiefly by changes in alveolar ventilation, to a lesser extent by the other mechanisms involved in the regulation of neutrality in the body (9); second, changes in rate of metabolism of the cells of the center, possibly accounting for changes in 'irritability' of the center and for nervous influences upon it; third, changes in rate of removal of metabolites from the center, chiefly through changes in the vessels supplying it, also through vasomotor responses, both sets of influences antagonizing chemical changes in the blood. The end result is a delicate balance between external and internal stimuli, a balance which can be disturbed either from without (through changes in the blood) or from within (through changes in rate of production or removal of metabolites) with the same result, namely, a change in concentration of stimulant material within the center and a corresponding respiratory response. We do not propose to amplify the term *Chemical Stimulant Material*. As far as the general conception is concerned it makes no difference whether one calls it CO_2 tension, oxygen tension, acid-base balance, or total acidity."

The "vasomotor responses" referred to are the general, systemic vasomotor reactions dependent on the vasomotor center of the medulla. The vasomotor changes in the cerebral vessels are not considered. The subject is well presented and discussed; such a discussion of an important and intricate mechanism naturally brings up various controversial points. It is to be deplored that editorial restrictions kept the author from presenting his data and protocols more at length, for the essence of the "scientific method" is the presentation of data so completely that the reader may draw his own conclusions without relying on the discussion of the author.

COBB, Boston.

PREVENTION OF MENTAL AND NERVOUS DISEASES. BERNARD SACHS, J. Nerv. & Ment. Dis. **68**:355 (Oct.) 1928.

In order to prevent nervous and mental disorders, there must be sound heredity, proper care of the mother during pregnancy, care and skill at the delivery of the child, sensible parental guidance through the first years of life, rational school training, careful supervision during adolescent years and the avoidance of sexual and alcoholic excesses. Members of families with a history of definite mental diseases should not marry, and the intermarriage of two unhealthy strains is positively to be condemned. Worry, neglect, emotional disturbance, malnutrition and renal diseases in the pregnant mother predispose to disease in the child. The mother should be protected from every form of infectious disease while pregnant. Improper obstetric methods at the time of delivery and particularly tedious labor are often followed by infantile cerebral palsies. Haste in these cases is better than delay. There should be isolation for one month in the case of anterior poliomyelitis and epidemic encephalitis to prevent the extension of these conditions in childhood. As syphilitic involvement of the cerebral spinal fluid may be found in the first year after the initial infection, one can start earlier with antisyphilitic treatment. Certain forms of progressive muscular atrophy can be stayed by early orthopedic measures and physiotherapeutic procedures.

In children with neurotic disturbances, a complete change of environment will, nine times out of ten, be the chief corrective. A child copies readily the nervous symptoms of an hysterical mother. If he can be placed under the influence of sober minded, intelligent persons for a period of months, or in a special school with normal children, under some discipline, the treatment is more likely to be successful. Underlying the mental hygiene movement is the desire to prevent mental diseases in later life by the early correction of these abnormalities. Obses-

sions in childhood are often due to some emotional experience, with subsequent development of habit.

Adolescence is a time when both sexes are apt to have mild mental disorders with moodiness and sense of inferiority. One must not be too indulgent with incipient neurotic persons or make much fuss over very slight peculiarities. Self-abuse in either sex should be frowned on and not treated with indifference. Children of psychotic stock should be brought into company with entirely normal children, especially in the kindergarten and early primary school.

Dementia praecox develops in the adolescent period when the first great strain in the struggle for existence is manifested. The adolescent is then often pushed by his own ambition and that of his parent beyond his own physical and mental endurance. Even the more generally regarded unfavorable mental disorders of the paranoid variety can often be held in abeyance by discussion and the kindly interest of the physician.

HART, Greenwich, Conn.

EXPERIMENTAL STUDIES OF EPILEPTIFORM CONVULSIONS. M. IRVING SPARKS, Arch. internat. de pharm. et de therap. **33**:460, 1927.

This is a thorough piece of experimental work on dogs. The cortex was exposed for the electrical experiments and the cortex around the cruciata sulcus was stimulated with the faradic current. Drugs were also used as convulsants: Thujone was used intravenously on sixteen dogs, and phenol and strychnine were given to cats both subcutaneously and by direct application to the cortex. Of especial interest is the author's observation that both clonic and tonic movements can be produced at levels lower than the cortex. This controverts the recent work of Dandy who believed that clonic convulsions were always cortical in origin.

In summary, the author states, in effect:

1. Electrical stimulation with weaker currents produces clonic, with stronger tonic movements. Sometimes a bilateral clonic seizure can be produced which starts after the electrodes are removed.

2. The electrical irritability of the brain is increased by the ketone thujone, and low blood sugar level; decreased by phenobarbital, and unaffected by morphine.

3. Thujone produces typical epileptiform convulsions when given intravenously. Susceptibility to it is increased by low blood sugar content unaffected by morphia, and decreased by phenobarbital and iso-amylethyl barbituric acid.

4. In doses slightly larger than those used therapeutically the only effect of phenobarbital is to reduce the responsiveness of the nervous system to the convulsant used. Subcutaneously, sodium phenobarbital exerts this effect in from twenty to forty minutes, while as a late result there occurs a peculiar ataxia and muscular weakness. Intravenously this form of the drug acts in from two to three minutes.

5. Iso-amylethyl barbituric acid is equally efficacious with phenobarbital in depressing thujone convulsions. It gives promise of being useful in clinical epilepsy.

6. Loss of one motor cortex changes the type of response and raises the minimal convulsant dose of thujone. Clonic movements, however, still occur bilaterally.

7. Loss of both motor cortices increases the spastic phase of a convulsion but clonic movements are usually present also.

8. Phenol applied locally to the brain produces a distinctive tonico-clonic response which requires a sensory stimulus to precipitate it.

9. Subcutaneous injection of strychnine produces a tonic response and increased irritability. Local applications to the brain produced no noticeable sensory changes but a peculiar tonico-clonic type of response similar to that produced by phenol could be elicited.

10. Tonic and clonic convulsions can both be produced at various levels of the neural axis and their production in the cortex is discussed.

COBB, Boston.

THE CELL-FREE ZONE AT THE POINT OF ENTRANCE OF THE CRANIAL NERVES INTO THE BRAIN IN VERTEBRATE EMBRYOS. A CONTRIBUTION TO THE COMPARATIVE HISTOGENESIS OF THE CRANIAL NERVE TRUNKS AND THE OBERSTEINER-REDLICH AREA. LEO ALEXANDER, J. f. Psychol. u. Neurol. **36**:350, 1928.

Alexander investigated the areas of entrance of the nerves in selachians in various embryonal stages, and the relation of the cell-free zone at the point of entrance of the nerves to the marginal layer in the brain of the embryo. Following this, he studied the histogenesis of the Obersteiner-Redlich area in man, and the comparative anatomy of this zone in fully developed brains. Particular attention was given to the rhombencephalic cranial nerves. The entire study is summarized as follows: At the points of entrance of the various rhombencephalic nerves there is found a zone which is completely devoid of cells. This cell-free portion of the anlage of the nerve, however, is not the anlage of the actual trunk of the peripheral nerve in the sense of His, but is the anlage of the Obersteiner-Redlich area which contains little or no myelin. The nerve trunk differentiates from the cell-containing proximal portion of the originally uniform cell-containing nerve plate, as the former runs distalward from the Obersteiner-Redlich area. Phylogenetically, the Obersteiner-Redlich area of the various rhombencephalic nerves of selachians is peculiar and corresponds ontogenetically to the cell-free zone at the points of entrance of the corresponding cranial nerves into the brain. Identical conditions are observed in man in the rhombencephalic cranial nerves, as well as in the spinal cord; in the latter the Obersteiner-Redlich zone of the entering posterior nerve roots has also a cell-free zone anlage. The Obersteiner-Redlich zone of the posterior nerve roots of the spinal cord of man, when subjected to examination by means of polarized light, showed a peculiar appearing myelin in that it seemed as if it were completely broken down. This appearance, however, was not due to any peculiar staining property of the cord but to an actual absence of doubly refractive lipoids in the area examined. The axis cylinders appeared naked, embedded in the zona marginalis gliae and covered only by their axolemma. In the sections examined, the Obersteiner-Redlich area showed marked individual variations which could be divided into various chief types and into transitional forms. Thus, there were found some in which there was a complete interruption, in others a thinning, and in still others an admixture of a partial interruption and partial thinning of the myelin sheath.

KESCHNER, New York.

NERVE FIBERS IN THE PITUITARY OF A RABBIT. MARGARET M. CROLL, J. Physiol. **66**:316 (Nov.) 1928.

The author tried a number of methods of staining, many of them being entirely unsuccessful and many giving only partially satisfactory results. The most successful of the methods used was the following modification of Ranson's silver-pyridine technic: the blood vessels of a rabbit were washed out with Ringer's solution, which was injected into the aorta after all the lower vessels had been tied off. One hundred and sixty cubic centimeters of ammonia alcohol (100 per cent alcohol with 1 per cent ammonia) was then injected. The pituitary was removed and cut in halves; the anterior lobe was cut through again, and the pieces were fixed for twenty-four hours in ammonia alcohol. After they had been washed in distilled water, they were transferred to pyridine for twenty-four hours, and washed for another twenty-four hours in many changes of distilled water. The pieces were then placed in the dark in a large quantity of a 2 per cent solution of silver nitrate at 35 C. for seven days, rinsed in distilled water and reduced for sixty hours in a 4 per cent solution of pyrogallic acid in 5 per cent commercial solution of formaldehyde. They were washed, taken quickly through the alcohols, embedded rapidly in paraffin and cut at 18 microns, and in one or two cases counterstained with safranine or methylene blue (methylthionine chloride, U. S. P.). This modification gave extremely good results in the case of the pars intermedia; in some cases parts of the anterior lobe also showed black fibers.

Croll finds that the pars intermedia shows numerous thin black fibers running between the cells and sometimes ending in knots. They are considered to be nonmyelinated nerve fibers which are not vasomotor in function, as the intermedia in the rabbit is poorly supplied with blood vessels. They do not resemble connective tissue, and appear constantly in sections stained by Ranson's method. Nonmedullated nerve fibers are present in the pars anterior. They do not occur in such numbers or so regularly as in the intermedia. Owing to the great wealth of neuroglia fibers in the pars nervosa the identification of nerve fibers in many areas is difficult. They are, however, abundant in the vicinity of the vessels and at the junction of the nervosa and the intermedia.

ALPERS, Philadelphia.

FURTHER EXPERIMENTS UPON THE TRANSPLANTATION OF EMBRYONIC SPINAL-CORD SEGMENTS. S. R. DETWILER, J. Exper. Zool. **52**:351 (Jan. 5) 1929.

Replacement of the second, third and fourth segments of the embryonic spinal cord of *Amblystoma* by the fourth, fifth and sixth segments of another embryo is accompanied by the following results: The fourth segment, which replaces the normal second, undergoes a slight hyperplasia (about 6 per cent). The fifth segment, which replaces the normal third, undergoes a hyperplasia of about 20 per cent. The sixth segment, which replaces the normal fourth, undergoes a hyperplasia of from 25 to 30 per cent. The failure of the fourth segment to undergo a greater cellular increase is attributed to the fact that it is substituted for a region of the cord which has been shown to possess a greater inherent potentiality for high proliferation than the succeeding segments. The factors which bring about the normal high proliferation in the first and second segments are apparently specific to this region and appear to be unaffected by alterations in the position of these segments; whereas in succeeding segments studied the degree of final cellular proliferation appears to depend largely on the position which they occupy with respect to the spinal cord as a whole. The purely local conditions which rigidly govern cell production in the first two segments seem to extend in part into the region of the third segment. It is indicated that below the second segment the final production of cells in any given segment is not entirely limited by local inherent potentialities for development, but falls more under the influence of general gradients.

A relatively lower cell production occurs in the first segment when followed directly by the fourth than under normal conditions. It is suggested that the second segment normally affects development in the first, particularly in the dorsal regions. This same force is operating in succeeding segments, but to a much more diminished extent. The rates of cellular proliferation occurring in the spinal cord, in the stages observed, are not influenced by extraspinal conditions. The mesoderm appears to play no part. The final number of cells which proliferate and differentiate appears to be determined by a variety of interacting forces which lie purely within the central nervous system.

WYMAN, Boston.

THE IMPORTANCE OF EXOGENOUS FACTORS IN THE ETIOLOGY OF SCHIZOPHRENIC DISEASE. F. ENGELMANN, Arch. f. Psychiat. **84**:588 (Aug.) 1928.

The author reports the results of a study of 123 schizophrenic patients taken as they were admitted to the clinic. The purpose of the study was to discover the importance of exogenous factors in the causation of the psychoses. Under the term exogenous factors the author understands not only physical but also psychic traumas. In twenty-eight cases, exogenous factors of this type played a real part in bringing about the psychosis. In fourteen, these factors were in the form of psychic trauma (in some, apparently isolated occurrences; in others, chronically repeated injurious experiences). In six others the psychic traumas were assisted by physical factors (menstruation, puerperium, etc.). In four cases, the main precipitating factor seemed to be some injury of the skull, and in the last four, the psychosis was apparently brought on by some other organic cerebral disturbance (encephalitis, intoxication, infectious disease, etc.).

The author comments on the literature on the subject and the importance of his observations. He admits that in all cases in which an exogenous factor seemed to be the sole cause of a psychosis there was, in all probability, some predisposition, either constitutional or acquired early in life. He thinks, however, that when the exogenous moment is of particular importance, as in the twenty-eight cases reported, one must consider it possible that the patient with the predisposition could have gone through life without the development of mental disease if it were not for this last precipitating factor. From a pragmatic point of view, however, one can regard this exogenous factor as the real cause of the disease.

In summing up the results of his investigations, the author comes to the following theoretical considerations: there must be special brain systems (not necessarily anatomically localizable) which are especially related to the type of reaction met with in schizophrenia. These systems may be constitutionally or otherwise more vulnerable than usual. It is possible that the so-called schizoid person has this type of predisposition. It is also possible, however, that infectious, endocrine or other toxic agents may affect these systems acutely and thus bring about a schizophrenic form of reaction.

MALAMUD, Foxborough, Mass.

THE DEVELOPMENT OF THE SPINAL CORD IN AMBLYSTOMA EMBRYOS FOLLOWING UNILATERAL MYOTOMECTOMY. S. R. DETWILER, J. Exper. Zool. **52**:325 (Jan. 5) 1929.

Complete removal of the right sixth, seventh and eighth somites of *Amblystoma* embryos in the tailbud stage results in a total absence of axial musculature, although the ventrolateral muscle sheet may be filled in from muscle buds of the adjacent segments. Study of the spinal cord shows that the proliferation of cells on the demuscularized side is as great as on the normal side. This bears out former conclusions that the proliferation of cells in the motor areas of the cord is not influenced by the presence of differentiating or functioning musculature. The extent of proliferation of primary spinal motor neurons must be conditioned by influences residing within the central nervous system. Although disturbances in the surrounding mesoderm may influence development of the cord in early stages, there is no indication that after the medullary tube is closed, the surrounding structures have any influence on its morphology.

Spinal ganglia have been found to segment and differentiate in the complete absence of axial musculature, but in all cases some axial cartilage is present. It may be that a developmental influence has already been impressed by the segmented somites on the ganglionic crest cells, so that, following their removal, this influence continues to act. Segmentation of the motor roots has been found to take place in the complete absence of sensory ganglia and adjacent myotomes. Their outgrowth is not dependent on influences exerted by the differentiation and early contraction of the muscle. The motor roots on the operated side in all cases are smaller than their counterparts. This is apparently due to atrophy resulting from lack of function, and not to any failure of the neuroblasts to differentiate. In cases when motor roots are present and the corresponding ganglion is absent, the motor root is supplied with sheath cells which have migrated down from the unsegmented crest cells. The absence of spinal ganglia in cases with complete somitic excision is not due to the result of the excision of normal crest cells. Control experiments in which attempts were made to remove completely the crest cells in corresponding stages were always followed by completely developed spinal ganglia.

WYMAN, Boston.

NOTE ON HISTOLOGIC TECHNIC (METHOD FOR STAINING MYELIN FIBERS AND GLIA FIBERS. V. M. BUSCAINO, Riv. di patol. nerv. **33**:486, 1928.

The author has tried to elaborate a method of staining myelin fibers in serial sections. The method proposed is a modification of that of Spielmeyer; blocks of tissue are embedded in paraffin, with previous passage through fluids, which leave

unaltered the substances that are essential to the fixation of hematoxylin. The blocks are fixed in pure acetone, either directly or after formaldehyde followed by a short washing. The tissue is left for about forty-eight hours in the acetone which is changed a few times according to the volume of the blocks. These are then passed into petroleum ether which in the process of embedding has the same action as xylene. The detailed technic is:

(*a*) Fix blocks directly in acetone or pass through acetone blocks that have previously been fixed in 10 per cent of commercial solution of formaldehyde.

(*b*) Place in petroleum ether for twenty-four hours or more.

(*c*) Petroleum ether and paraffin.

(*d*) Embed in paraffin.

(*e*) Section at 10 microns.

(*f*) Deparaffinate the sections.

(*g*) Stain with Spielmeyer's method for myelin fibers for deparaffinated sections.

To obtain impregnation of glia fibrils the author proposes the following method:

(*a*) Fix blocks of tissue in absolute alcohol.

(*b*) Embed in paraffin.

(*c*) Stain by the method of Spielmeyer for myelin fibers with deparaffinated sections. With this method the glia fibers and the protoplasmic glia are well impregnated.

FERRARO, New York.

SURGICAL INDICATIONS IN MENINGO-ENCEPHALITIS WITH CIRCUMSCRIBED CORTICAL CONVULSIONS. WERNER BUDDE and WILLI CROME, J. f. Psychol. u. Neurol. **37**:64, 1928.

The authors report the cases of two previously healthy middle aged men who, without discoverable cause, suddenly developed tonic-clonic twitchings of half the face and tongue. At first these came on at great intervals, but within a few days they became more frequent and finally almost continuous. The twitchings were associated with a gradually increasing paralysis of the muscles involved, with tonic contractions in the homolateral upper extremity. There was no disturbance of consciousness during the attacks. One of the patients also gave a history that eleven years before, after a severe excitement, he developed a convulsion during which he lost consciousness, bit his tongue and vomited; he had a similar attack fourteen days later. Both patients were neuropathic. Except for increased deep reflexes on the side involved, the neurologic examination gave negative results. The cerebrospinal fluid was normal. For some days preceding the operation, one of the patients had a subnormal temperature. Both patients were treated with various antiepileptic remedies with no relief. Owing to the great restlessness, insomnia and inability to take nourishment, the patients were subjected to trepanation on the tenth and twelfth days (respectively) after the onset. In both cases the operation revealed a circumscribed serous leptomeningitis on the contralateral centers for the involved muscles. The operation was followed by a rapid disappearance of all symptoms. The authors had a similar experience six years before in a similar case. In all these cases the entire symptom-complex resembled Kojewnikoff's epilepsia partialis continua. The etiology of this condition has not as yet been definitely determined although it is believed by some to bear some relationship to encephalitis or poliomyelitis. The good results following trepanation would seem to justify early operation in similar cases.

KESCHNER, New York.

POSTPARTUM SCHIZOPHRENIAS. G. ZILBOORG, J. Nerv. & Ment. Dis. **68**:370 (Oct.) 1928.

According to the author's psychoanalytic formulations, based apparently on two cases of postpartum schizophrenia, the girl normally leaves the auto-erotic stage, in which she desires to possess a penis, by wishing instead to possess a man (the

father), identifying herself with her mother in order to have a child by him. While pregnant, the expectant mother "invests the future offspring with libidinous energy." The child, therefore, becomes the mother, the sexual partner, the sexual partner's penis and the woman's father, not to mention the father's penis and its anal equivalent.

This, apparently, is not enough, for the author also "identifies" the child with its mother's ego, or as a "narcissistic addition to the ego." To the author's mind, feces, penis and child are all in some way to be identified with one another; but when the woman begins to perceive the movement of the fetus, the child loses its anal significance. The author concedes that all these identifications bring about an extremely complicated set of conscious reactions toward the child. As he expresses it, "Upon the future child, the whole gamut of emotions is played, from the narcissistic self-delight to anal sadistic hatred, and from depressive ambivalence to triumphant masochistic self-sacrifice." During the act of parturition, women pass through an "orgy of libidinous joy." Unfortunately, the patients in the cases described could not renounce their claim for a penis or accept the child as a compensation. Both were frigid, which means that they identified themselves as men and needed none. At times, they manifested a passionate desire for intercourse which meant the attempt to reincorporate the penis. Thus, fundamentally, they suffered with a castration complex of the revenge type, an oedipus situation and father identification. All this throws much light on postpartum schizophrenia.

HART, Greenwich, Conn.

INFANTILE CEREBRAL PALSIES. LUCIANO MAGNI, Riv. di patol. nerv. **33**:231 (March-April) 1928.

This paper is a complete and thorough review covering the interesting and difficult field of infantile cerebral palsies, or — using the more adequate term of Brissaud — infantile encephalopathies. Discussing at first various tendencies, the author points out the fallacies of a one-sided approach to this complex subject. He reviews briefly the etiologic factors of infantile encephalopathies: abiotrophies, syphilis, hereditary predisposition, etc. With regard to syphilis the author judiciously keeps himself to the golden middle course where most probably the truth is found. He then discusses the pathologic changes of infantile encephalopathies: tuberous sclerosis, Strümpell's diffuse sclerosis and cerebral lesions with a tendency to affect the basal ganglia — Wilson's hepatolenticular degeneration, and Vogt's status marmoratus and status dysmyelinizatus.

The most interesting part of the review is that in which clinical forms and neurologic syndromes of infantile encephalopathies are reviewed. Instead of the usual narrow classification into two syndromes, infantile hemiplegia and cerebral diplegia, Magni describes in a masterly way the syndromes of double athetosis, pyramidostriatal syndrome, infantile pseudobulbar palsy, mesencephalostriatal syndrome of decerebrate rigidity, chorea congenita, etc. A brief but complete synthetic review of the pathophysiologic data obtained in later years concerning extrapyramidal disturbances makes this clinical part of the paper most helpful in obtaining an orderly orientation in this field of neuropediatry.

Any worker in such institutions as schools for the feebleminded, hospitals for epileptics and neurologic departments of children's hospitals will find an important synthesis in this paper.

YAKOVLEV, Palmer, Mass.

PSYCHOSES IN CORTICAL ENCEPHALITIS. PAUL SCHILDER, J. f. Psychol. u. Neurol. **37**:293, 1928.

This contribution is based on three personally observed cases of encephalitis and a review of the literature on this subject. The cases were infectious in origin but not of the epidemic variety. The encephalitic process was not strictly limited to the cortex. From this study Schilder concludes that encephalitic changes in the brain give rise to exogenous types of psychoses: 1. Organic delirium and

comatose states (severe cortical encephalitis). 2. Occupational deliria, as in epidemic encephalitis when the cortex participates in the pathologic process. Amentia is rare. 3. Relatively slight damage to the brain with cortical involvement produces a delirium which may merge into hallucinations. 4. The latter resemble closely the paranoid-hallucinatory forms of dementia paralytica, cases that have been subjected to malarial therapy and in which the cortex is also involved. 5. In contrast to chorea minor, the encephalitic cases are characterized by the presence of cortical symptoms and by a less frequent occurrence of profound confusional states. 6. The psychoses in chronic encephalitis, in contrast to the cases described in this paper, show better insight and greater accessibility, and although hallucinations are present the hallucinatory character of the psychosis is not very evident. 7. Cortical lesions due to encephalitis or to conditions simulating it (Quincke's edema) may give rise to psychoses of the schizophrenic type. It would seem, then, that the psychoses per se do not differ very much from the ordinary postinfectious type of psychosis; it is only the presence of the associated cortical symptoms that justifies their being considered a peculiar type of psychosis.

KESCHNER, New York.

INVERTED POSITION IN CHILDREN'S DRAWINGS: REPORT OF TWO CASES. GERALD H. J. PEARSON, J. Nerv. & Ment. Dis. **68**:449 (Nov.) 1928.

As Putnam has shown, an object perceived is projected on the retina as an inverted image, and this remains inverted in the visual cortex, for the upper and lower halves of the retina are represented in the upper and lower lips of the calcarine fissure. The reinversion of the image, so that the object is seen right side up, is a learning process, which probably occurs very early in life when the child is unable to indicate his difficulties to other people. Traces of this period of learning can be found in drawings of children of the preschool stage.

In two cases, one a girl, aged 6, with an intelligence quotient of 60, and the other a boy, aged 5 years and 5 months, with an intelligence quotient of 97, the majority of objects were drawn upside down. When the object was placed upside down, the first child drew it right side up. In both children the motor coordination was poor.

Stern has shown that only very young children can recognize pictures as easily when upside down as when right side up. Spiller reported the case of a boy, aged 8, who saw everything upside down after an attack of measles. In adults, cases of a temporary disturbance of this sort have been reported by authors after head injury.

HART, Greenwich, Conn.

EPIDEMIC ENCEPHALITIS. L. SYLLABA and K. HENNER, Arch. Int. Med. **42**:151 (Aug.) 1928.

This rather lengthy report deals with about 1,000 cases seen in Czechoslovakia during the years 1919 to 1925. The authors consider it to be a disease entirely distinct from influenza, and view it as a complete clinical, pathologic and anatomic entity. In addition to the commoner and classic signs of epidemic encephalitis, they note a number of rare observations: rhythmic clonic contractions, seen in six cases; medial facial spasm (which they believe to be the first reported); bradykinesia, seen in three cases, and consisting of slow rhythmic movements of the limbs. They also found every combination of loss and preservation of the pupillary reactions; the most frequent of these is the reverse Argyll Robertson pupil.

On the side of treatment, they discuss the virus vaccine of Levaditi, used intrathecally. They are inclined to believe it had some effect, but admit its true value is impossible of determination. Especially do they remark that they have not made control attempts with nonspecific protein. Sodium cacodylate was used with some subjective improvement. The final comments are on the knowledge which encephalitis appears to be giving on the extrapyramidal system.

ANDERSON, Kansas City, Mo.

FUNICULAR MYELITIS. O. ALBRECHT, J. f. Psychol. u. Neurol. **37**:13, 1928.

Albrecht reports a case of funicular myelitis associated with pernicious anemia in which microscopic examination of the nervous system showed, in addition to the usual lesions of funicular myelitis, marked involvement of the blood vessels of the cord, especially of the vasocorona. He believes that this and similar cases give an important clue to the pathogenesis of funicular myelitis. It would seem that the entire pathologic process in the cord is due to an involvement of the vessels in the vasocorona, as a result of which there occurs a marked nutritional disturbance in the affected structures of the cord. The author, however, has no explanation to offer why the posterior columns are a site of predilection for the lesions, although in his opinion this is no hindrance to the theory that the nerve changes in this disease are due to nutritional disturbances. He is absolutely opposed to the theory of a hypothetic toxin as the causative agent of the pathologic process in the nervous system.

KESCHNER, New York.

CERTAIN STATES OF CURABLE INSANITY, SIMULATING SCHIZOPHRENIA. SANTIN CARLOS ROSSI, Encéphale **23**:501 (June) 1928.

The author reviews the long recognized fact that since psychiatric diagnoses are made more on the basis of descriptive symptomatology than on that of pathologic anatomy, there has been a tendency to diagnose a case as dementia praecox and then consider the diagnosis in error if the patient recovers. The term "schizophrenia" is so broad as to include a multiplicity of symptoms. In fact, all symptoms that are possible literally cannot be described. It follows that in some cases there will be relative preservation of the affective life and therefore that various combinations of the schizophrenic and the cyclothymic reactions actually do occur. This is so true that the term schizomania is justified as indicating both types of elements. These elements differ in acuteness of onset, preservation of affective reactions, chronicity and, above all, curability. Some such relationship exists as that between meningitis and meningismus. Three cases are described in elaboration of the view.

ANDERSON, Kansas City, Mo.

THE INSULA IN MAN AND IN ANIMALS. MAXIMILIAN ROSE, J. f. Psychol. u. Neurol. **38**:467, 1928.

In this contribution Rose describes the architectonic structure of the island of Reil in *Chiroptera, Insectivora, Rodentia, Prosimiae, Simiae* and *Homo*. After a review of the literature he devotes 119 pages to a description of his personal investigations carried on in the Kaiser Wilhelm Institute for Brain Research in Berlin. The next nineteen pages are devoted to ontogenetic, phylogenetic and comparative considerations of this important part of the brain. The concluding four pages are devoted to a discussion of the functions of the island. The paper contains 114 illustrations and 38 microphotographs. For obvious reasons, these illustrations cannot be reproduced in an abstract and without them no intelligible abstract is possible. Any one interested in the subject must read the original paper to be able to appreciate this monumental piece of research.

KESCHNER, New York.

ENCEPHALITIS PERIAXIALIS DIFFUSA — TYPE SCHILDER. WALTER M. KRAUS and ARTHUR WEIL, Encéphale **23**:775 (Nov.) 1928.

After a brief review of cases in the literature and a discussion of the terminology of the condition, the writers describe a case of their own. This patient, aged 34, when first seen in 1911, was thought to have multiple neuritis. He was then seen at intervals for the next thirteen years, his condition being variously labeled multiple sclerosis and hysteria. The course was progressive, with the development of tremors, hemianalgesia, aphasia, convulsions, a Babinski sign, hypertonia, etc. At the time of death, in 1924, extensive areas of demyelinization with preserved

cylinders were found in the subcortical white matter, and also in the medulla and pons. These demyelinized zones were replaced by a heavy neuroglial network; vessels were surrounded by large spaces filled with small round cells and plasma cells. The process is regarded as a secondary reaction to the demyelinization.

ANDERSON, Kansas City, Mo.

TRANSPLANTATION OF ANTERIOR-LIMB MESODERM FROM AMBLYSTOMA EMBRYOS IN THE SLIT-BLASTOPORE STAGE. S. R. DETWILER, J. Exper. Zool. **52**:315 (Jan. 5) 1929.

The technic of the grafting of tissue in *Amblystoma* embryos earlier than the medullary plate stage is described. The results show not only that the forelimb mesoderm in the embryos of *Amblystoma punctatum* is segregated and localized before the medullary plate appears, but that it has already become polarized along its anteroposterior axis, so that, when the mesoderm is inverted and grafted into slightly older embryos, limbs with reversed asymmetry (disharmonic) differentiate. Just how early the limb mesoderm is segregated and its anteroposterior polarization determined can be ascertained only by grafting in still younger stages. The present experiments point to the view that the polarization of the anteroposterior axis may take place as early as limb mesoderm itself becomes segregated and determined.

WYMAN, Boston.

TREATMENT FOR EPILEPTIC MANIFESTATIONS BY PHENYLETHYL MALONYLUREA. JACQUES LEYRITZ, Encéphale **23**:787 (Nov.) 1928.

This work is based on twenty-one patients with essential epilepsy who had been under observation since 1922, and had been started on phenobarbital in May, 1926. The total number of attacks suffered by the twenty-one in these years were as follows: 1922, 1,091; 1923, 1,174; 1924, 1,211; 1925, 1,212; 1926, 706, and 1927, 161. This drop in response to the new medication is certainly graphic. The effective doses ranged from 0.1 to 0.4 Gm. In addition to definite action on the epileptic attacks, it exerted a most beneficial influence on the psychic equivalents — several cases being cited in support of this. Some evidence is also adduced that mental deterioration is less noticeable under its use.

ANDERSON, Kansas City, Mo.

THE EFFECT OF TEMPERATURE ON THE MELANOPHORES OF FISHES. DIETRICH C. SMITH, J. Exper. Zool. **52**:183 (Nov. 5) 1928.

The melanophores of *Fundulus heteroclitus* are capable of responding directly to changes in the temperature of their environment, as is shown by the effect of heat and cold on the melanophores of isolated scales and denervated portions of the tail and trunk. Melanophores denervated in any way always contract to high temperatures and always expand to low temperatures. Innervated melanophores in the tail always react to warmth and cold qualitatively as denervated melanophores do. Innervated melanophores of the trunk always react to warmth and cold in the reverse way from denervated melanophores.

WYMAN, Boston.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Dec. 20, 1928

H. C. Solomon, M.D., *President, in the Chair*

The Conversion of a Psychosis Into Hysteria. L. E. Emerson, Ph.D.

This case is of particular interest because of the change in the predominating symptoms from psychotic to hysteric, under known conditions. When I first saw the patient, on Jan. 4, 1928, she had a psychosis. She was loud in her denunciation of her mother's employer, who, she said, had encouraged her husband to desert her and was responsible for his failure to return. (About three weeks previously her husband had deserted her and she had not been able to find him since, though she had appealed to the police.) She talked of nothing else. It seemed as though ideas of reference were focused on one innocent person. At this time, she spoke of no other symptoms and seemed not to be troubled in any other way. The observations that led me to consider her psychotic were: (1) the vehemence and viciousness of her feelings of fury; (2) the groundlessness of her accusations; (3) the tendency to race on, enlarging on her grievances and entering into innumerable ramifications, and (4) her not uttering a word of reproach for her husband. A further reason for considering the patient psychotic was that she lacked insight. She was unaware of her perilous condition and of its causes and meanings.

It was found that she had been in the hospital before, in November, 1927, because it was a question whether she had tuberculosis, but nothing was found proving the presence of this disease. It was noted at this time that she had a "psychopathic personality."

The patient, aged 26, was born in Belgium, and came to the United States when she was about 14. She had been educated in convents and was devoutly religious. She had been married about two years when I saw her, and although quite unhappy during her married life, she was "passionately devoted" to her husband and said, "I will not live if I cannot see him again." The first night that he was away, she tramped the streets, going from one police station to another, trying to get help to search for him. She had been all along the water front, taking his picture to shipping agents.

The patient was seen four times, at irregular intervals, in January, and not again until April 20. In the meantime, she had secured a position as a mother's helper. This was the reason why she had not come regularly.

After the patient had vented her rage, she became more communicative as to the treatment she had suffered from her husband. After telling of numerous perversions, she divulged the fact that he had committed an abortion on her, in her ignorance, against her will, and with this confession she began to see more clearly that her desperate feelings had their origin in his treatment of her. Thus, the danger of a complete psychosis was averted.

In April, a friend telephoned that the patient had been taken to the emergency ward of a hospital as a result of having been knocked down on the street by her husband. It was found that she had a strained back but no other injuries.

When I saw the patient again, on May 1, she informed me that she had lost her case for separate support in the lower court, but that the state was taking it to a higher court. At this time, I noticed that she was more willing to let her husband go, since he had so vilified her in court, even accusing her of being immoral. On May 9, the patient's mother came to the clinic and said that the case for assault and battery had been heard the day before, and that the husband had been fined $25; he appealed. She said that her daughter had left home the night before, saying that she would never be seen again. The mother was sure

that the daughter had committed suicide. She asked the police to watch for her. The patient returned home the next day.

On May 16, I had a long interview with the patient. I thought it might be possible to help her to react normally to the situation, but I knew it would take a long time, and advised her admission to the hospital. She came into the hospital on July 5. She complained of a slight bleeding from the rectum. She said that she had bled there before when her husband had had intercourse with her there. She is quiet and comfortable and has made no trouble of any kind since the operation.

Now, conditions were right for the conversion of her psychosis into hysteria, and this proceeded to take place.

Just before the patient was to be discharged from the hospital, the mother came to me and said that she and her husband wished to have their daughter committed. She said that her daughter could not go back to the French Home, as the latter had no room for her and that she could not come with them because she had threatened her father's life with a knife. This discouraged me. Up to this time there had been no observed sign of hysteria; everything had pointed to a psychosis. Therefore, I consulted with Dr. J. B. Ayer and told him the facts, so far as they were known to me then, and said that I agreed with the mother that the patient should be committed. On the basis of the facts as I told them Dr. Ayer agreed with me, but when we saw the patient together, she was so quiet, amenable and undisturbed that he changed his mind, and because she seemed to be underweight and anemic, he advised that she be sent to Chickering House to be built up.

According to the Social Service notes on the case, the social service worker, on July 30, took the patient to Chickering House in her car. The mother came with clothes for the patient and helped her to dress. When the patient was ready, the worker was astonished to find that the patient could not stand. She trembled all over and lost her balance. The head nurse assured the worker that this would pass off after a little use of her legs. It was difficult to get the patient into the automobile.

On July 31, the house physician, Dr. Benton of Dedham, saw the patient. He diagnosed her case as hysteria. The patient had to leave. A Mrs. Sonnenberg then received the patient. It was necessary to have the patient carried downstairs to the car, and carried up the steps at Mrs. Sonnenberg's home.

On August 8, Mrs. Sonnenberg said that the patient was happy and contented. Mrs. Sonnenberg was worried about the patient's condition because she seemed to have unassociated movements of her body whenever she tried to walk or stand. Her feet and legs were cold. The patient made no trouble and Mrs. Sonnenberg was glad to do all she could for her.

On August 21, the patient was admitted to the Adams Nervine Asylum. On this date, she was still unable to walk a step or to bear her weight without losing her balance. She complained of pain in the right leg and side and a lack of circulation in both legs. Dr. E. B. Lane now stated that the case was hysteria.

One must think that a patient is in a rather serious condition when she attacks her father with a knife. It is, perhaps, much less dangerous if a girl attacks her father with a carving knife than if a boy attacks his mother with a hatchet. Boys are much more likely to do something; and I think that when one sees the transformation and sees both sides of it, it looks so reasonable and every motive is so clear, that one is likely to read into the patient's mind the clearness that he himself possesses, whereas actually it does not exist. It is a transformation of one state of mind into another state of mind. William James speaks of insane delusions and ordinary delusions, which are false beliefs. After all, we are all subject to delusions, for we all have false beliefs; but there is a world of difference between an insane delusion and an ordinary delusion. This girl was in such a state of mind that she was suffering from an insane delusion, in the sense that she had no consciousness at first that all her feelings were not caused by her mother's employer, who became the object toward which she directed them. She was without insight. Lack of insight is the only way in which one can, perhaps, define insanity.

THE RELATION BETWEEN THE SIZE OF THE VENTRICLES AND THE CLINICAL IMPROVEMENT IN TREATED CASES OF PARESIS. DR. H. C. SOLOMON and DR. ARTHUR BERK.

Thirty-two patients in whom we were able to determine the size of the ventricles by the injection of air were studied. They showed that there was no relation between the clinical improvement following treatment and the size of the ventricles. But, on the other hand, there was a definite correlation between ventricular size and the improvement shown by psychometric examination. This led to the further demonstration that there is no definite correlation between the change in the psychometric observations and the patient's ability to care for himself in the community. That is, patients whose intelligence quotient greatly increased after treatment were unable to leave the hospital, and patients whose intelligence quotient went downward were able to go out and make good. Finally, there was no relationship between the clinical syndrome presented by the patient and the size of the ventricles, nor was there any relationship between the duration of the psychosis and the size of the ventricles.

DISCUSSION

DR. D. J. MACPHERSON: I remarked to Dr. Solomon, relative to the increase or decrease in the intelligence quotient, that the important fact seemed to be the intelligence quotient at termination of treatment. If the patient started with 90 and lost 21, he still had more than the man who started with 40 and gained 6.

THE INFLUENCE OF MALARIAL TREATMENT ON THE HISTOLOGIC CHANGES IN PARESIS. DR. RICHARD B. WILSON.

The numerous reports on the treatment for paresis by inoculation with malaria offer convincing evidence that the improvement to be secured through its use is far greater than one can expect to obtain with any other method; thus, a knowledge of the histologic changes following malarial treatment becomes of considerable significance. In an effort to determine what these changes are, histologic studies have been made in fifty-six cases. For the present purpose, it seems advisable to consider only forty-five of these, apart from the atypical ones and those terminating after three years. In the latter group, the changes cannot be correlated with those found in cases terminating earlier, and it seems doubtful that any influence of the malarial treatment is manifested at so late a period.

The series of changes following the inoculation with malaria may be divided into three periods: (1) a period of exacerbation, (2) a period of restoration and (3) a period of recrudescence.

The period of exacerbation covered an interval from the beginning of treatment to six weeks after the termination of the fever. This period was observed in twenty cases. At this time, typical but unusually severe histologic changes of paresis were found; there was a strong meningeal infiltration in which macrophages were numerous; the cortical perivascular infiltration was intense and diffuse. The degree of the proliferation of the microglia cells tended to go parallel with that of the infiltration. The walls of blood vessels and the microglia cells gave an unusually strong iron reaction. This increased intensity of the inflammatory reaction was in accord with most previous observations. There were, nevertheless, some discrepancies. Sträussler and Koskinas, Freeman and Nakamura reported a predominance of lymphocytes in the cortical perivascular spaces; in these cases, there was a predominance of plasma cells throughout. Some also reported a reaction similar to that found in cerebral syphilis; that is, a reaction characterized by a strong mesodermal proliferation with the formation of nonsyphilitic granulomas and even typical miliary gummas. Freeman emphasized this, and Sträussler and Koskinas found miliary gummas shortly after the end of treatment, but in only two cases of their series of thirty-five. Kirschbaum, in twenty-two cases, did not find such a reaction, nor was it seen in these cases. Toward the end of this period there was an increased intensity of all reactions; but the perivascular

infiltration became gradually less diffuse, so that the infiltration predominated on the arterioles and venules. Spirochetes were found in one case.

Following this there was a period of restoration. This was observed in nearly three fourths of Kirschbaum's cases. Shortly after six weeks, there began a gradual diminution of all reactions and a concomitant improvement in the appearance of the cortical architecture; the decreasing perivascular infiltration was confined almost exclusively to the arterioles and venules. In corresponding degree, there was a reduction of the proliferation of the glia cells and of iron in the walls of the blood vessels. No spirochetes were found. Only one case terminating during a remission was secured. Here the arrangement of the cortical layers and of the nerve cells was good, although the evident loss of ganglion cells indicated that considerable destruction had been effected. The perivascular infiltration was almost negligible, only occasional lymphocytes and plasma cells being found in the perivascular spaces. The microglia and macroglia cells were not proliferated. It was rather striking, in contrast with this, that there should be a moderate amount of iron in the walls of the blood vessels.

In a smaller group of cases (seven of twenty-four) after six months a new flare-up of inflammation occurred: a period of recrudescence. Among four cases terminating between six and nine months after treatment, one case showed such a reaction, and among eleven terminating between one and two and a half years after treatment, there were six. In one of these, the inflammatory reaction was intense, but the amount of iron in the walls of the blood vessels was small. The other six cases may be described as a group, since the changes in all were, in principle, the same. Throughout the brain there was a slight perivascular infiltration, but the outstanding feature was the scattered foci in which the thickened walls of the blood vessels were intensely infiltrated, often with the cells breaking through into the nerve tissue itself. First, there was proliferation of the mesodermal elements and organization, so that here the cells of infiltration lay in layers embedded in a mesenchymal mesh. With increasing mesodermal proliferation there was a narrowing of the lumen of the blood vessel, and soon there was complete obliteration. Occasionally, central pale areas were found in this mesodermal network, which probably indicated beginning necrosis. When this process went still further, typical miliary gummas, with necrotic centers and giant cells, were found. Of six cases in which the search was made spirochetes were found in five.

With the exception of this group, spirochetes were found only once (one case in twenty-nine). It is well known how seldom spirochetes have been found after malarial treatment. This would seem to indicate that the malarial treatment has effected a considerable destruction of the spirochetes. Forster reported finding spirochetes in material removed during life. In one case, the material was removed seven weeks after the end of treatment; this, it is seen, was toward the end of the period of exacerbation of all reactions, and it is likely that the destruction had not been completed at the time of examination. In the other case, the material was removed six months after treatment; a time when, as already pointed out, a secondary flare of inflammation often occurs. These results all lead to the conclusion that the treatment has destroyed or greatly reduced the spirochetes and that after six months they may regain their virulence and, proliferating, cause a recrudescence of inflammation.

It is generally accepted that, in untreated cases, little correlation is possible between the severity of the clinical course and the intensity of the inflammatory reaction. It seems probable that the inflammatory reaction is the expression of the body's effort at defense, whereas the severity of the clinical course depends on the physiologic state of the parenchymatous tissue. Thus, it would follow that, in cases with slight inflammatory changes, which yet terminate fatally, there have been such severe alterations of the parenchymatous elements that functional recovery is no longer possible.

No definite parallelism has been found between the histologic and the serologic changes, although in cases with histologic evidence of activity the serologic reac-

tions have remained stationary or have become worse; in the other cases, they have shown a slight tendency toward improvement. One serologic reaction that appears to be of considerable significance is that with mastic. Kirschbaum and Kaltenback found that in cases showing clinical improvement the reaction with mastic becomes weaker; with a reappearance of symptoms, the reaction becomes stronger again. The reaction with mastic, when compared with the histologic changes shows the same tendencies; that is, during the period of exacerbation it becomes stronger and with histologic improvement it becomes weaker; in the group of cases showing a recrudescence of inflammation, it is even stronger than in the cases in which the patients died during the treatment. It was pointed out that during and shortly after treatment macrophages are rather numerous, as indicated by Walter's bromide method, with the iron in the cortical blood vessels. In these cases a definite parallelism exists, which tends to substantiate Spatz' contention that the iron pigment present in paresis is of hematogenous origin, and that it indicates an increased permeability of the blood vessels. No such relationship exists in cases other than paresis; not even in those with a high permeability is iron to be found in the cortical blood vessels, or at least, only a rare granule of iron pigment. Zalka and Lehoczhy examined 100 patients and were unable to find iron in the cortical blood vessels. The next question was to determine whether the choroid plexus plays a rôle in the passage of substances into the cerebrospinal fluid. In none of the cases examined could specific changes be found nor any iron pigment detected. If it is accepted that in cases not paresis, increased permeability for bromide indicates an injury either physical or physiologic, then the injury to this barrier in paresis must be different in type or degree, since in these cases the barrier permits, in addition to the passage of bromide, that of blood pigment; furthermore, such an injury seems to be confined to the vessels of the brain, the choroid plexus remaining uninvolved.

Earlier, I reported that the iron in the microglia is proportionate to the amount in the walls of the blood vessels. This assertion was based on the study of treated cases only. Since then, three untreated cases have been studied. In all of these, in spite of an intense iron reaction in the walls of the blood vessels, the amount of iron in the microglia cells was disproportionately small; in two cases, almost negligible. It would seem, therefore, that the malarial treatment causes an increased transportation of the iron pigment from the walls of the blood vessels to the microglia cells. This observation was made also by Sträussler and Koskinas. It is interesting that Hoff and Silverstein found an increased opsonic index for streptococci, staphylococci and colon bacilli in the blood and spinal fluid of patients during treatment. Their studies offer suggestive but not conclusive evidence that there is an increased phagocytosis for spirochetes. It seems possible that the presence of macrophages in the meninges may be a histologic expression of this increased phagocytosis.

The question of the permeability of the barrier between the blood and the cerebrospinal fluid has received considerable discussion in recent years. Weil and Kaĭka obtained a positive reaction of hemolysin in the cerebrospinal fluid in most cases of paresis, and regarded this as an indication of an increased permeability. Pötzl, and later Horn, found that it disappears after treatment, usually after two months. Walter, using the bromide test, found an increased permeability in paresis. Malamud and others confirmed these results, finding an increased permeability during treatment and with clinical improvement, a decrease; later, concomitant with or preceding the return of clinical symptoms, the permeability again increases. All these studies seem to indicate that the barrier between the blood and the cerebrospinal fluid has been injured, as a result of which toxic substances, or even substances normally present in the blood, pass more readily into the cerebrospinal fluid. An effort has been made to determine what relationship the permeability of this barrier has or may have to the histologic changes. No definite correlation has been found between the degree of permeability and the intensity of the inflammatory reaction. This is also true for the proliferation of glia cells, which tends to run parallel with the amount of infiltration.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 21, 1928

N. W. WINKELMAN, M.D., *President, in the Chair*

A CASE OF CEREBROSPINAL SYPHILIS WITH MARKED CEREBELLAR ATAXIA. DR. M. A. BURNS.

Clinical History.—P. L., aged 39, a clerk, complained of an unsteady gait, swaying from side to side, some slight difficulty in speech and marked ataxia. There was no history of any nervous or mental disease in the family. The patient had been well until the age of 19 when people began to notice that he did not walk properly and that he stumbled considerably; this was more pronounced in the dark. The condition grew slowly worse until about two or three years before presentation when it became so aggravated that he was obliged to take a vacation for fourteen weeks; after that he improved slightly, but the condition has gradually been getting worse. At the age of 14 his tonsils were removed. At the age of 15 he was struck on the head by a baseball; he was unconscious for several hours and was confined to bed for four or five weeks; the ball hit him just above the nose, but he does not think that his skull was fractured by the blow. The patient is married, and has one child who is living and well. The wife never had any miscarriages. For some months past, he has had attacks of severe pain in the stomach. These attacks come on suddenly and cause him to roll about in anguish for several hours, the pain then leaving as rapidly as it appears. He has never vomited during these attacks. His father had similar gastric pains.

Examination.—There was a marked Romberg sign; the gait was markedly ataxic, with some swaying to the right side. Eyes: The movements were normal; there was no ptosis or nystagmus; the pupils were regular, of normal size, equal and reacted promptly to light and in accommodation. There were no cranial nerve palsies. Upper Extremities: Muscular power was good; there was no atrophy, tremor, adiadokokinesis, astereognosis or dysmetria. The biceps and triceps reflexes were about normal and equal on the two sides. Lower Extremities: Muscle power was good and there was no atrophy. The heel-to-knee test showed marked ataxia. The patellar reflexes were exaggerated but equal on the two sides; the achilles reflexes were about normal; there was no evidence of clonus or Babinski sign. The senses of vibration, position, light touch and pain were normal over the whole body.

Special Tests.—Lumbar puncture showed no increase in pressure; the fluid cell count was 6 or 7 per cubic millimeter. The Wassermann reaction of the spinal fluid was strongly positive. The Wassermann reaction of the blood was positive. Examination of the eyes gave negative results. Roentgen examination showed that the sella was larger than average but had perfectly formed clinoids, and there was nothing to suggest erosion or destruction. The vascular markings were pronounced, but there was no localizing increase.

Diagnosis.—A diagnosis of cerebrospinal syphilis with marked cerebellar ataxia was made.

ENCAPSULATED CEREBRAL HEMORRHAGE. DR. J. WILSE ROBINSON, JR.

The patient, a white man, aged 66, complained of headache and malaise in the fall of 1924. He was sent to Florida; while fishing he had a stroke which affected the whole left side. He was admitted to the Philadelphia General Hospital on May 9 to the neurologic service of Dr. J. W. McConnell, twenty-three days after the onset of the condition. When admitted he was a well developed man in a semiconscious condition. He responded when spoken to, but his answers were irrelevant. He was oriented as to time and place but did not know his name. He complained of a paralytic condition of the left arm, the leg and lower part of the face.

Examination revealed weakness of the lower part of the left side of the face. The tongue was protruded in the midline without tremor. The eyes showed only the changes of advanced age. The chest was hyperresonant, and there were many crackling râles in both bases. The heart was enlarged to the left, and the liver protruded below the costal margin. The reflexes on the right were normal while those on the left were exaggerated. The abdominal reflexes were present. A Babinski sign and clonus were obtainable on the left and were also questionably present on the right. The left extremities were spastic and paralyzed. The temperature and pulse and respiratory rates were never abnormal. The blood pressure was not recorded. The laboratory observations were all within normal limits.

The patient died on June 2, forty-seven days after the onset. The diagnosis made was of right cerebral thrombosis and chronic myocarditis.

At autopsy the interesting general observations were: hypertrophy and myocardial degeneration of the heart with secondary coronary sclerosis; congestion of the liver and spleen, and chronic glomerular nephritis.

The neuropathologic examination showed a brain which was normal in size, shape and weight. The hemispheres presented a moderate edema. Grossly, the uncut brain showed nothing but a fibrosis of the membranes and a marked arteriosclerosis. Section of the brain in a horizontal plane showed a hemorrhage occupying the right ganglion area. It was roughly oval and measured 5.5 by 2.5 cm. It had destroyed the posterior portion of the internal capsule, the lenticular nucleus, the external capsule and the island of Reil, and had invaded the anterior portion of the interior capsule. It was older than the usual hemorrhage, as it was surrounded by a membrane and was apparently becoming organized. The gross diagnosis was arteriosclerosis and old, right, encapsulated cerebral hemorrhage.

Microscopically, the hemorrhage had undergone degeneration with pigment formation at the margins. At its immediate edge there was a ring of large phagocytic cells within a faint meshwork of fibrous tissue. As one passed toward normal brain tissue the phagocytic cells became smaller and had a tendency to group around blood vessels where they had the appearance of lymphocytes. The brain tissue around the hemorrhage showed a "status spongiosus."

Oppenheim, in his "Textbook of Nervous Diseases," stated that apoplectic cysts may be found after three or four weeks. The adjacent cerebral substance forms, by proliferation of the glia and by new formation of connective tissue, a kind of a capsule around the focus. The contents of the capsule are more and more absorbed until there remains only a cavity filled with serous fluid. It is seldom that any complete cicatrization takes place.

Buzzard and Greenfield, in their work "Pathology of the Nervous System," stated that, early, the seat of the hemorrhage is occupied by a red clot which is easily separated from the surrounding tissues. These are infiltrated and softer than usual. Later, the clot and the walls undergo changes. The clot shrinks, remaining red in the center and growing yellow at the periphery. After a lapse of time, the coagulum becomes completely absorbed, and its place is taken either by proliferated scar tissue or by a quantity of more or less blood-stained fluid. Changes in the wall are taking place during this time, namely, the destruction of the nerve elements and the proliferation of the neuroglial tissue. The newly formed glial substance either forms the lining membrane of the cavity or binds the walls together. The final results may be either a large single cavity, a multilocular cavity or a linear scar. These relics can scarcely be distinguished from other lesions of similar antiquity.

A brief study of the current literature on this subject brings out two points: (1) This is a rare condition. (2) Brain hemorrhage, even though extensive, does not produce sudden death. In 1906, Spiller reported thirteen cases of extensive hemorrhage of the brain, in which death occurred in from five hours to two months after the onset. The patient who lived for two months showed a right spastic hemiplegia and an aphasia. He was operated on a month and a half after the onset, and a left subdural hemorrhage was found. He died two weeks later, and a hemorrhage measuring 7 by 2.5 cm. was found in the left lenticular nucleus and the internal capsule. The clot appeared to have been of long duration and was partly encapsu-

lated. He concluded that a diagnosis of hemorrhage of the brain was not justified in cases of sudden death.

Thomas, Spillsburg and Malony concluded from many cases of sudden death that cerebral hemorrhage is not a cause of sudden death; while agreeing with them, Oppenheim said that medullary hemorrhage will cause sudden death.

Cadwalader found that cerebral hemorrhages are apt to be large. In only four of the twenty-four cases reported in 1911 did the hemorrhagic area measure less than 4 cm. in its broadest diameter. He concluded that large hemorrhages are always fatal, and that it is probable that all hemorrhages may be fatal. He also did not believe that small hemorrhages will cause sudden death.

Winkelman and Ekel, in an analysis of thirty cases of proved hemorrhage in which the time of death and onset were definitely known, believed that cerebral hemorrhage does not cause immediate death, that is, in less than one hour, even with rupture into the ventricles or within the pons.

Douglas, in 1925, reported a hemorrhage of four years' duration which was encapsulated and cystic. There was an area of yellowish pigment surrounding the cyst which was proved to be blood pigment. The patient died of an extensive hemorrhage on the opposite side of the brain.

I believe from these studies that a clot in the brain may be walled off if the patient survives long enough, but while cerebral hemorrhages do not cause sudden death, they are usually fatal in a short time and therefore sufficient time does not elapse for the formation of a capsule. This, I believe, explains the rarity of this condition.

A Case Showing Multiple Cerebellar Lesions. Dr. Helena E. Riggs.

History.—Wm. G., a colored man, aged 47, was admitted to the Philadelphia General Hospital on the service of Dr. Charles Burr on Dec. 2, 1927, and died on Dec. 24, 1927. In 1926, he had vertigo and was treated for high blood pressure. In April, 1927, he showed signs of mild cardiac decompensation. In June, 1927, he had severe vertigo with temporary blindness; these attacks occurred sometimes once a day and sometimes only once every two weeks; vision was approximately normal between the attacks. He also complained of dull boring occipital headaches. There was nausea and vomiting, and umbilical pain. The gait was staggering, and he tended to walk to the right. He also complained of double vision at this time.

Examination.—The blood pressure was 250 systolic and 170 diastolic. The heart was greatly hypertrophied; a presystolic murmur was heard at the apex; ventricular extrasystoles were present, and there was marked peripheral arteriosclerosis.

No lesions of the cranial nerves were detected. There was exaggeration of both patellar tendon reflexes; all other reflexes were normal; there were no abnormal reflexes. No paralysis or paresis was present, and there was no change in sensation.

Coordination.—Incoordination was marked in the left arm and leg. Slight incoordination was present on the right side. There was no asynergia. Adiadokokinesis was present in the left hand. The patient staggered to the left in walking.

Laboratory Data.—Serologic tests were entirely negative. The blood chemistry was normal.

Course.—On the third day after admission, there were: weakness of the external recti, lateral nystagmoid movements and slurring of the speech. On the fifth day after admission, the spinal fluid pressure was 50 mm. of mercury. The spinal fluid showed: a heavy trace of globulin; colloidal gold curve, 2333221100. The serologic tests were negative. The ocular fundi showed papillitis with patches of exudate. Bárány tests gave normal results.

The intraspinal pressure remained high in spite of repeated spinal drainages. The headaches were relieved by spinal drainage. The patient was found dead in bed twenty-two days after admission.

Autopsy Observations.—Observations showed hypertrophy of the heart, atheroma of the aorta and chronic nephritis.

Brain.—There was severe sclerosis of the basal vessels with tortuosity. The right vertebral penetrated deeply into the under surface of the right cerebellar

hemisphere and into the right cerebellar peduncle. The vessels also invaginated the right surface of the medulla. In the right cerebellar hemisphere there were two areas of recent hemorrhage. These were about 2 cm. in diameter. One lay anterior and adjacent to the restiform body. The older hemorrhage was in the left lateral hemisphere.

A Case of Gumma of the Brain with Onset of Symptoms Following Trauma: Operative Removal with Recovery. Dr. Temple Fay.

The case is of interest because of the definite history of severe head trauma just prior to the onset of symptoms. The negative Wassermann reactions of the blood and spinal fluid before operation and the prompt positive serologic observations after exploration and removal of the tumor are especially noteworthy.

History.—C. R., aged 38, a painter, was admitted to the service of Dr. Arnett at the Episcopal Hospital on Oct. 27, 1928, complaining of severe temporoparietal headache associated with mental confusion. Five weeks prior to admittance to the hospital he had been struck on the head by a 40-foot ladder. He was unconscious and was taken to a hospital, where he remained for nine hours. He then was able to drive his car home, but became confused, so that he lost his way and did not reach home for several hours. He was helped into his home by a friend, who thought the patient was intoxicated. Two days later the wife noted that he did not understand what was said to him, and that he had difficulty in finding the proper words when speaking. He became confused and drowsy.

The past medical and family histories were essentially unimportant. The patient's occupation brought him into close contact with lead, and he had had lead colic at one time. He was left-handed in his work, but close questioning brought out that he was right-handed by birth.

Examination.—The neurologic examination, by Dr. George Wilson, indicated that a partial word deafness was present and some jargon aphasia, with bilateral Babinski sign, more marked on the right. There were no other focal symptoms.

Roentgen examination gave negative results. The spinal fluid pressure was 8 mm. of mercury (normal). The Wassermann reactions of the spinal fluid and blood were negative. The cell count of the spinal fluid was 37 lymphocytes.

A diagnosis of lead encephalitis, hemorrhage or syphilis was made by Dr. Wilson, and an exploration over the left sylvian area was advised.

Operation.—I made the usual osteoplastic flap and uncovered the left temporoparietal lobes. Just below the sylvian fissure, and about the central part of the first temporal convolution, was a firm, white tumor, adherent to the dura and infiltrating into the brain itself. The tumor was about the size of a small plum and was situated deeply within the brain substance. It was not encapsulated, requiring removal by dissection.

Course.—The patient made an uneventful recovery and was discharged from the hospital on the tenth day. He still has some word deafness, but in all other respects he is symptom-free. He is receiving antisyphilitic treatment and is greatly improved.

One week after the removal of the tumor the Wassermann reaction of the spinal fluid was found to be four plus and of the blood two plus.

Pathologic studies of the tumor were reported by Dr. Winkelman as follows: "Section shows an area of cortex in which there is marked pathology. At the margin one sees cortex over which the meninges are thickened, fibrotic and filled with cells. There are many lymphocytes and plasma cells. Penetration into the cortex occurs, and there is perivascular infiltration within the cortex. The small blood vessels show marked wall thickening with swelling, and reduplication of the lining cells. The vessels are all markedly distended. The tumor itself is made up of a dense fibrous tissue background with areas of necroses within, and about them islands of lymphocytes. An occasional giant cell is met with. Microscopic Diagnosis—Gumma of the brain."

The case is of interest from three standpoints: the rapid onset of symptoms following a definite head injury; the negative Wassermann reaction of the spinal

fluid before operation, and the situation of the tumor in what proved to be Wernicke's zone. The auditory aphasia was the most marked symptom in the case, but this was not complete, and slight jargon aphasia was also associated with the slight motor manifestations. The appearance of a positive Wassermann reaction after the operation in both the blood and the spinal fluid was thought also to be of interest.

DISCUSSION

DR. ALFRED GORDON: In view of the fact that you found trouble near the tip of the temporal lobe, I should like to ask whether there were any symptoms regarding the senses of smell or taste?

DR. C. A. PATTEN: Has Dr. Fay any explanation for the curious serologic observations? Was there a sharply localized infection with negative serology at first, which, because of the operation, became scattered and distinctly positive later?

DR. TEMPLE FAY: Regarding Dr. Gordon's question, I have tried to localize the tumor in the sketch. It was below the sylvian fissure and anterior to the angular gyrus area. The sense of smell was tested and found to be normal, but the sense of taste was not tested.

Dr. Patten's question was asked Dr. White of the Episcopal Hospital staff, who responded that the test was conducted as usual, and was checked in the City Laboratory as well. He could not explain the result, but was inclined to resent the suggestion that the laboratory might have made a mistake. We have to accept as a fact that the Wassermann reaction was at first negative, and after the operation became two plus in the blood and four plus in the spinal fluid.

MASSIVE SUBCORTICAL HEMORRHAGE SURROUNDING A BRAIN TUMOR: OPERATION WITH RECOVERY. DR. TEMPLE FAY and DR. NICHOLAS GOTTEN.

The case is of unusual interest because a recent hemorrhage dissected free and outlined an old tumor mass which was found after the large subcortical clot had been removed from the right hemisphere. Both lesions were removed, the patient making an interesting recovery.

History.—E. G., a colored woman, aged 25, was admitted to the Philadelphia General Hospital on Oct. 12, 1928, on the service of Dr. D. J. McCarthy, complaining of paralysis of the left side of the body. The symptoms had begun ten days before admission, and were characterized by weakness, malaise, numbness and tingling of the left arm and leg. These symptoms continued until October 12, when she awoke to find complete paralysis of the left arm and leg. She had noted severe frontal headache for the twenty-four hours prior to the onset of the paralysis.

Three years before, the patient had had vertigo for three days, followed by a generalized convulsion with loss of consciousness. She was in bed for three days and recovered without further symptoms. She had had two convulsions limited to the left arm and leg, without loss of consciousness, since the onset of the present illness.

Examination.—On admission, with the exception of the paralysis of the left arm and leg, the general examination gave negative results.

Neurologic Examination.—The biceps, triceps, patellar and abdominal reflexes were diminished; the knee jerk was increased on the right and normal on the left; there was no Babinski sign or clonus. Speech was normal. Paralysis was marked in the left leg, and was complete in the left arm and hand. Sensation to heat, cold and vibration was normal. The blood pressure was 106 systolic and 62 diastolic. The spinal fluid pressure was 16 mm. of mercury; the fluid was clear and no cells were reported. The Wassermann reactions of the blood and spinal fluid were negative.

Course.—Seven days after admission, the patient developed focal epilepsy, limited to the left side of the face and the left arm. The following day she became comatose, and the jacksonian attacks in the face and arm became almost continuous.

A diagnosis of right parietal neoplasm was made, and an exploratory craniotomy was recommended.

Operation.—An exploratory craniotomy (Dr. Temple Fay) was done on Oct. 23, 1928. "When the dura was exposed, two unusual things were noted. (1) a dark, hemorrhagic injection of the subarachnoid space in the superior central convolution, (2) a pale, avascular zone in the convolution, anterior to the dusky area, situated close to the falx at the midline. An incision of the arachnoid over the dusky area showed no blood. Opening the cortex at a depth of 0.5 cm. disclosed an oily semi-liquid clot, about 3 ounces in amount, extending down to the level of the corpus callosum and toward the internal capsule. Evacuation of the clot by suction revealed the outline of a small, hard tumor mass involving the upper layers of the cortex anteriorly. The clot had dissected and isolated two thirds of the tumor mass from below. The tumor was dissected free and removed. The clot having been evacuated there was no indication of the point of origin of bleeding. The patient was so stuporous during the initial steps of the operation that no anesthetic was necessary."

Following the operation, the patient made an uneventful recovery, and within three weeks was able to move both her left arm and leg and to walk with assistance.

Pathologic Report (Dr. Winkelman).—"Microscopic examination of the tissue shows the following: There is a small (not larger than ¾ inch [19.05 mm.] in diameter), comparatively old hemorrhage which has been completely walled off by a fibrous capsule in which there are present phagocytic cells, in small numbers, most of which contain a brownish pigment. The cortex in the immediate neighborhood shows degeneration, with glial proliferation, and numerous large cells containing pigment. In one part of the section, the surrounding blood vessels show coloring with pigment-carrying cells. Adjacent to this is a bit of very recent hemorrhage with marked reaction around it, but no walling off. Microscopic diagnosis: (1) old hemorrhage encapsulated, (2) recent hemorrhage with reaction."

The case is of interest in that a subcortical hemorrhage if accessible can be removed by suction, and, even though extensive, offers a chance for recovery. The observations of an old hemorrhagic lesion in the midst of the recent clot was surprising. The mass was small and firm, and though perhaps not a true tumor, was a distinct lesion resembling a tumor. Its character was disclosed later by Dr. Winkelman.

DISCUSSION

Dr. Temple Fay: It has been four weeks since the operation, and there is still marked weakness. Frankly, I felt at the time I saw the patient that it was almost hopeless to do anything. Yet here was a focal lesion demanding intervention. A dusky area on the upper precentral convolution gave the impression that there was something under the surface. I am surprised to find that the woman can use her arm and leg. The area was large (one could put four fingers in the dissection the clot had made). I did not detect the tumor mass until I had taken out the clot. Apparently it had been an old hemorrhage followed by a recent one.

This case is rather surprising, and the fact that the patient survived brings up the point whether it is worth while making an attempt to remove the clot in cases of massive subcortical hemorrhage. Although, formerly, I would have said in this type of case that it is not worth while, the fact that the patient recovered proves that perhaps it is.

Dr. N. W. Winkelman: This specimen has caused a great deal of discussion. Dr. Fay removed a small nodule which he thought to be a tumor and sent it to the laboratory. There was found a rather old hemorrhage, about ¾ inch (19.05 mm.) in diameter, which showed the most marked capsule I have seen about a hemorrhage. The meshes of the capsule were not like those found in the dura, but were distinctly distended. The products of degeneration were being taken away. To any one looking at it under the microscope there could be no doubt that it was undergoing encapsulation. Then the question came up, is that in the nature of a tumor? It may be that a year or so from now Dr. Fay may operate again and find a tumor; I cannot say that the material received was a tumor.

I think that Dr. Spiller's comment is to the point. If these cases are fatal, as Dr. Robinson has shown, then surgical intervention is certainly indicated if it can do what it has done for the patient here.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 4, 1929

GEORGE H. KIRBY, M.D., *President, in the Chair*

AMAUROSIS IN EPIDEMIC ENCEPHALITIS. DR. IRVING T. SANDS.

A white woman, aged 37, with an unimportant family and personal history, was admitted to the hospital on April 16, 1927, complaining of loss of vision, excruciating frontal headache and cough. On March 16, she had given birth to a normal child. The puerperium was uneventful. On April 2, 1927, she attended a wedding and spent the greater part of the evening in the company of a friend who was suffering from a cold in the head and who was coughing and sneezing throughout the evening. On the following day, the patient complained of headache and went to bed, with a temperature of 102 F. that subsided in three days. On April 7, she complained of dimness of vision, and later complained of seeing peculiar objects, distorted figures, animals, etc., which were present even on closing the eyes. On April 13, she complained of smoke before her eyes, and she could not recognize the members of her family.

On admission to the hospital, the right pupil was larger than the left, and both reacted sluggishly to light. The eyegrounds showed blurring of the margins of the disks and retinal congestion. The left eyeball was rotated to the left. There were no other neurologic disturbances. The heart and lungs were normal, except for the presence of many râles throughout the entire chest. A chronic tonsillitis was present. The nose and sinuses were normal. The blood pressure was 130 systolic and 75 diastolic. Urinalysis gave negative results. The blood chemistry was normal. A blood count showed 14,500 white cells, with the other elements normal. The Wassermann reaction of the blood was negative. The spinal fluid was under increased pressure; it showed ten cells, 65 mg. of sugar, and negative Wassermann and colloidal gold reactions. The temperature was 99 F., the respiratory rate, 22, and the pulse rate, 70.

On April 18, she was unable to distinguish between day and night. She then showed a papilledema of 2 diopters in each eye, with retinal hemorrhages. X-ray examination of the sinuses showed no pathologic changes. There was an irregular calcification in the pineal gland.

On April 20, the patient developed hypesthesia and hypalgesia in the right trigeminal area, and there was a left supranuclear facial paresis. The pupils did not react to light but reacted sluggishly in accommodation. She complained of numbness in the right side of the tongue and in the right angle of the mouth. She had a tonic and clonic convulsion, lasting for about four minutes, associated with loss of consciousness and foaming at the mouth.

On April 23, she became confused and disoriented for several hours. There was loss of conjugate elevation of the eyes, and a horizontal nystagmus was present on looking to the right. She was nauseated and vomited. There was hyperexcitability of both semicircular canals. The diagnosis of epidemic encephalitis was made, and typhoid vaccines, intravenous injections of hypertonic solutions of dextrose and spinal drainage were instituted.

On April 30, the patient began to perceive light, and from then on continued to improve. The papilledema receded, and the hemorrhages were absorbed. Optic atrophy then supervened. She was discharged from the hospital on June 17. She had been seen regularly every month. At the time of presentation she had an optic atrophy. Vision was 5/200 in each eye. She showed loss of associated movements, masked facies and slight tremor. She was able, however, to look after her household, and to read the headlines in the newspaper. The combination of papilledema, blindness, convulsions and optic atrophy made the case worthy of presentation. The beneficial effect of foreign protein therapy was apparent.

DISCUSSION

DR. S. P. GOODHART: Dr. Sands chose for his subject one of the most intricate, interesting and subtle of the innumerable symptoms which present themselves in epidemic encephalitis; his case presentation and its analysis are well chosen. The case illustrates some of the difficulties of differential diagnosis between tumor of the brain and encephalitis. The term "amaurosis" strictly speaking refers to loss of vision in which no objective observations are determined, and this distinction is made also by French writers. I do not think it is a desirable basis for distinction since, in early cases of retrobulbar neuritis, blindness without changes in the disks is frequently observed. As a matter of fact, the literature on encephalitis contains a number of instances of complete loss of vision without any objective observations in the visual apparatus. It seems not unlikely that some of these cases are due to retrobulbar pressure on the optic nerve and that others again are due to scattered lesions, including some in the visual cortex. Everyone knows, and the literature confirms what some have seen, blindness in encephalitis; especially early in the disease and without changes in the disks, but ending in recovery of vision. Of course, much more common is what Dr. Sands brought forth in his description of the case. In fact, one is no longer surprised to see even marked degrees of papilledema in epidemic encephalitis. The question as to the mechanism of origin as distinguished from that in tumor of the brain is of importance. Meinicke reminded one that the literature on influenza for the past thirty-five years contains histories of optic neuritis, and sought an explanation for it. It would not seem unlikely that with the congestion and local inflammatory reaction of sinus membranes, invasion of the optic sheaths by edema might readily occur. On the other hand, in many cases of epidemic encephalitis, the mechanism is doubtless the same as in tumor of the brain, that is, interference with the production and absorption of the cerebrospinal fluid—essentially a mechanical process. Walker, an ophthalmologist, in studying x-ray pictures of the sinuses and of the optic canals and foramina, called attention to the difference in size and shape of the skull in individual cases. In one of his cases he observed that there was a double optic neuritis, and with the recession of the inflammatory process the sight in one eye was recovered; while in the other, by reason of the secondary optic atrophy which followed, permanent blindness resulted. The case was one of epidemic encephalitis. Roentgenologic examination of the canals showed a great difference in the size of the canals and of the foramina of the two sides, and Walker concluded, with apparently good reason, that the secondary atrophy was due to compression of the nerve in its small canal and foramen. It is not unlikely that the early haziness of vision so often complained of is due to local pressure rather than to toxic reaction. Perhaps the most important feature that Dr. Sands' case illustrates is a possible recovery of vision even though severe optic neuritis has occurred.

DR. SMITH ELY JELLIFFE: The fact occurred to me that I heard no symptoms reported that were not well known in influenza. Practically every symptom that Dr. Sands reported has been reported in a large number of cases, ever since the epidemic of 1889, and Lichtenstern's large monograph gives a copious collection of them. Still, at the same time, while I am not for a moment doubting Dr. Sands' diagnosis, it seems to me that he did not present any evidence controverting the possibility of influenza.

There are certain items of interest. Although one is not certain about the epidemiology, ten hours is a little fast in any reported case that I remember for epidemic encephalitis infection to develop. Two days is the shortest time for contagion in the literature on epidemic encephalitis. Also, hemorrhagic retinitis is common in the amaurosis of influenza, whereas, so far as I know in the literature, hemorrhagic retinitis is comparatively rare in encephalitis. I think that Dr. Sands should have covered more fully the fate of the carrier of the infection; did her case go on to an encephalitic evolution?

Another point of interest is the report of the Argyll Robertson pupil. Loss of light reflex without involvement of accommodation has been more or less

widely found, but careful analysis has shown that the opposite is much more likely to occur in encephalitis, namely, disturbances of accommodation with preservation of light reflex. So-called cases of Argyll Robertson pupils have been reported in the history of encephalitis, but most of the observers who have followed them out have found that they were transitory; few cases are found in the literature, reported by those who have made a specific study of the matter, in which the Argyll Robertson pupil was a persistent observation in encephalitis. It may be temporary, but not persistent. Blum, in his interesting thesis, comes to this conclusion. On a priori grounds, however, there is no real reason why a true Argyll Robertson pupil may not be a permanent condition. Furthermore, I should like to learn from Dr. Sands whether there is any trace of parkinsonism in the patient at present, which would still further verify the diagnosis between epidemic encephalitis and influenza. That is by no means unimportant, from many points of view, and thus far he has not mentioned it.

Dr. Goodhart has so well covered the problem of the differentiation between tumor of the brain and encephalitis that I shall not go into that. I think the presentation which Dr. Sands has made does credit to himself.

Dr. Joshua H. Leiner: Did Dr. Sands find the sugar in the spinal fluid increased, to establish the question of a definite epidemic encephalitis? I did not hear him mention it.

After Dr. Foster Kennedy reported his cases of optic neuritis (I believe he spoke of thalamic syndromes), I reported three cases. In following up these cases of optic neuritis with thalamic involvement, I found that they occurred when the pyramidal tract and not the extrapyramidal system was involved.

Dr. J. W. Stephenson: Apropos of the differentiation between tumor of the brain and encephalitis with papilledema, it has been stated, and I have seen demonstrated, that the behavior of the spinal fluid is significant. As intimated by Dr. Goodhart, in encephalitis there is increased volume and decreased absorption of spinal fluid. Therefore, during the ingravesence of the encephalitis there will be an increase in the volume of fluid, manifested by the rapidity and volume of flow of spinal fluid from the lumbar puncture needle, which will reach its maximum during the greatest activity of the disease, and will subside with retrogression of the active process. As stated, I have seen this demonstrated in a proved case. In the case of tumor of the brain, which produces mechanical blocking, one may readily visualize a reversal of this behavior. Did Dr. Sands observe such a phenomenon in his case?

Dr. Sands: In reply to Dr. Jelliffe, I wish to state that I have investigated the condition of the carrier of the infection. She was in good health after getting over her mild cold. The patient now has a definite parkinsonian facies, loss of associated movements and tremor.

The sugar content of the spinal fluid varied between 65 and 90 mg.

The differential point mentioned by Dr. Stephenson was not known to me. I, therefore, did not pay attention to the rate of flow. But I never had any difficulty in obtaining spinal fluid from the patient; it always came out freely.

Submucous Resection as a Castration Symbol. Dr. C. P. Oberndorf.

Dr. Oberndorf first traced the well known physiologic connection between the nose and the female genitalia and gave examples from the literature of the symbolism of the nose for the male genital. He then presented the psychoanalysis in a case in which the nose actually replaced the penis as a sexual organ in attempts at copulation.

The case was that of a man, aged 24, who came for psychoanalysis after attempted suicide, which he felt was the only solution remaining to him after repeated unsuccessful attempts at coitus. His failure in copulation was caused by the fact that in each approach to a woman he was overwhelmed by a compulsive sniffling; with the onset of the sniffling, his erection disappeared. The patient

recalled an incident at the age of 5 when, trudging behind his mother on a rainy day, his nose came in contact with her skirts, and the perception of an odor reminiscent of feces caused him to have an erection. He subsequently made frequent attempts to repeat the pleasurable episode, although early he experienced a sense of guilt in connection with this act.

He was reared in an extremely rigid Catholic household and intended to prepare for the priesthood. The psychosexual religious conflict began before his first communion, at the age of 8, and continued with increasing intensity up to the time when he abandoned his clerical studies at the age of 19. During the course of the analysis the patient revealed unmistakable longings for copulation with his mother, which in turn he displaced on the sisters in the parochial school and the nuns of the church which he attended. He had steadfastly refused to believe in any form of conception other than the immaculate conception as recorded in the birth of Christ, and clung to the idea of birth per rectum until the age of 19. The sniffling turned out to be an equivalent for copulation with the mother and mother substitutes, and certain fantasies indicated with unusual clarity that coitus represented the desire for reentry of the patient's entire body into the mother's abdomen. When this sniffling, which had been a slight habit since the age of 11, began to thwart normal sexual activity, the patient reasoned that some obstruction in the nose interfered with the complete aeration of his blood; this in turn prevented the proper flow of blood to the genitalia. He therefore submitted to a submucous resection, but after the operation felt entirely hopeless and helpless, and the sniffling became intensely aggravated. The operation on the nose had the effect of an unsuccessful castration which left no alternative for the patient other than suicide, although the latter represented to him a mortal sin.

From a diagnostic standpoint, the patient presented symptoms sufficient to have warranted the classification of schizophrenia, depression of a manic-depressive type, compulsive neurosis or conversion hysteria. The analysis, which lasted more than two years, resulted in the cure of the symptom for which the patient came, a complete relief of the depression, and an adjustment of the clashing sexual and religious impulses. The treatment was undertaken without any interruption of the patient's occupation in which he has since achieved decided success.

DISCUSSION

DR. SMITH ELY JELLIFFE: Dr. Oberndorf's communication opens up so many vistas along which one might progress that one could certainly occupy as much time in the discussion as he did so profitably, but perhaps not so profitably. When he opened his discussion with a quotation from "Tristram Shandy" I was reminded of a recent statement that I read in Whitehead's "Science and the Modern World" —an interesting phrase that reverberated ideas which I had held and thoughts which I had had of my own—it goes something like this: "The pilgrim fathers of the scientific imagination of the present day are the Greek dramatists: Aeschylus, Sophocles and Euripides, and what they deal with under the symbol of fate the scientific imagination of the present day deals with under what we call the scientific law." Whitehead stated further that if one wants to find out what the scientific imagination of the century is really occupied with, one must go not to the scientists but to the literary people, particularly the dramatists and the poets; this is only a reaffirmation of what he said before. It is, therefore, comprehensible that Freud should have picked on the Oedipus complex as a law, because one knows how Euripides and Sophocles dealt with the Oedipus complex under the symbol of fate. Dr. Oberndorf has illustrated beautifully how the fate of breaking the Oedipus law can be analyzed more or less in terms of exact science, which exact science, or inexact science if you choose to call it so, has been termed psychoanalysis.

One thought that came to my mind as I heard the paper was the sexual significance of the organ of smell. Biologically, we all know something about it. In almost any man, the olfactory sense is of all the others practically the one

which predominates in the choice of the sex object. The cloaca, of most of the lower animals is both a vagina and a rectum, and, therefore, the combined object of the vagina and the rectum has been written into the phyletic history of the animal series in a definite form. From the comparative anatomic point of view the significance of the olfactory lobes as the oldest parts of the aftercoming cortex is well known. The oldest parts of the striatum and cortex are chiefly of olfactory origin. Smell is intimately associated with all the instinctual strivings of the human body. Some say that smell has become one of the subordinate senses of the body, but I do not believe it. It is only repressed in favor of the distance receptor mechanisms of sight and sound. This case shows what happens when the repression has become unsuccessful.

A second point which came to my mind is the enormous significance of the religious mechanisms to work out sublimation of these early instinctual cravings. I think that Dr. Oberndorf has well illustrated that, notwithstanding the fact that the submucous resection failed, the patient's history also showed there was a partial failure in the religious sublimation; that the religious sublimation did not work sufficiently well for this particular patient, although it works well for millions, if not billions, of the population of the present day.

Dr. Louis Hubert (by invitation): As a laryngologist, I have seen a number of cases of dysmenorrhea in which relief was obtained by the application of cocaine to certain regions of the nasal mucous membrane. In some of these cases, the relief is probably due to suggestion. One woman came into the Manhattan Eye, Ear, Nose and Throat Hospital, and her pain was relieved by cocaine. I did this once; when she came a second time I used sterile water, and the pain was also relieved, so I thought that the relief of the pain in this case at least was due to suggestion. Sex disturbances may cause certain symptoms within the nose, like the so-called vasomotor rhinitis type I have seen lately. Three months ago, a man from a mid-western town came to see me, complaining of nasal obstruction and watery nasal discharge, especially at night. He said that he had seen the most prominent specialists in Chicago and in his own town, and no one could help him. They treated him from an endocrine point of view, and they tried to test out whether he was sensitive to pollens, food proteins, etc. I went into his sexual history, which was fairly interesting. He said that while young he was sexually active; that he met a young French girl and had for about two years abnormal sexual intercourse with her; then he thought he would like to change his life and get married; he found that he was partially impotent; at night he would get a complete obstruction of his nose and a watery nasal discharge. The implication was that this nasal obstruction and watery discharge interfered with sexual intercourse, but I think that it was the other way around. Dr. Oberndorf would be able to explain the mechanism. I think that instead of getting an erection of the penis he got an erection of the turbinal of the nose, and then a watery discharge. The only way to find out if this discharge is watery and not due to a latent sinusitis is by having the fluid examined. I had it collected, and the examination showed it contained water and mucin, and no pus cells.

Dr. Oberndorf: In regard to what Dr. Jelliffe said, I have not really sufficiently stressed in the reading of my paper the extent to which the disintegration of the religious sublimation was responsible for this young man's neurosis, and I think that his complete break in faith was induced not only by the facts he learned concerning his mother, but also because his church-going father seduced each one of his young girl cousins as they came of age.

Corroborating Dr. Hubert's remarks, I have at present under analysis a patient in whom a profuse rhinorrhea will appear, as a complex indicator of seduction. If I say the word seduction to him, the nose will begin to run. He went to see a competent neurologist who, not knowing the patient was under analysis, told him he thought the rhinorrhea must be nervous and not to let any one operate on him.

THE PSYCHOLOGIC CONTENT OF EPILEPSY. DR. FRITZ WITTELS, Vienna, Austria. (By invitation.)

My aim is to draw attention to a fact which I have observed frequently enough in epilepsy to suggest a characteristic of this disease. This fact is connected with another one, already known, namely, that one can produce a seizure if one touches the chief complex, or one of the chief complexes of the patient. What I consider as new and as my own contribution, and would like to have controlled by other investigators, is this:

I could prove in the seizure, produced experimentally by touching the complex, not only that the seizure had a psychic content, but that I could uncover this content during the seizure. The psychic content always meant the peculiar sex life of the epileptic patient. Psychoanalysis emphasizes that the symptoms of neurotic patients represent their sex life. They have no other sex life, or the remaining part of their so-called sex life is at least considerably depreciated by the neurotic symptoms which are even more valuable to the patient than the manifest remaining portion of the sex life. It is interesting and new that one can prove that in an organic disease like epilepsy the sex life also is strictly connected with the symptoms of the disease. What I mean is not the frequently observed masturbation of epileptic patients during a spell, but unconscious sexual fantasies deeply repressed and appearing in the psychic content of the epileptic seizure.

Fedja, a Russian émigré, aged 40, a lawyer, lived with his father, aged 78, and a sister, more than modestly on a small income. The sister was married and had a child. She, her husband and the child occupied one room; the father and Fedja occupied a small adjoining room, and the maid, Natacha—a peasant brought along from Russia—slept in the kitchen. All six ate together in the kitchen. The patient was a socialist of the right wing before the revolution. After the revolution, the family, forced to leave the fatherland, wandered through Europe for four years before finally settling in Vienna. Two years before presentation, seizures began with absent-mindedness and distraction. Later, they turned into petit mal. In the midst of speech, the patient would stop, would not fall, but would sit or stand rigid, look around as if lost and begin a ruminating movement of his jaws. With his left hand, he produced a spasm known as the obstetric position. This attack would last a few minutes. The normal life would then continue, and he would know nothing of what had happened.

Fedja proved to be a fanatic. He refused to deal with bourgeois or with socialists; with bourgeois because they were guilty of the Russian revolution, with socialists because they did not know anything about "true socialism." It seems as if he alone knew what true socialism meant. He had had no sex life. Never in his life had he had normal intercourse. There was much masturbation until 1914. There was some mystic connection between this activity and the outbreak of war which seemed to bring an end to his masturbation. After the Russian revolution, he continued masturbation. Since he had become epileptic there had been no more masturbation. He suffered from erections, and poured cold water on his genitals to relieve pain. The patient attributed his disease to a connection with this sex misery, and believed he would get well if he had normal intercourse, but his principles forbade prostitutes. He had no social contacts outside his immediate family, so there was not much hope.

The father was a pedant, always grumpy and a tyrant in his home. He controlled his 40 year old son as if he were still a child. The mother died in Russia. Shortly before the seizures began, the father had an operation on the bladder. Before the operation, Fedja had to catheterize him for nearly a year. It is remarkable that Fedja had forgotten this activity, and when I learned it from another person and asked him directly he denied it.

He had an older brother, Alexis, who remained in Russia. This boy seemed to be a peculiar fellow—a wandering preacher on a theosophic basis. The Bolsheviks seemed to consider him harmless and left him alone.

Analysis of Dreams.—Dream 1: "Either I talked in a meeting and I was Babel, or I heard him talk, but I don't know what he said."

As an association to this dream he brought: "Babel's chief work is called 'The Woman and Socialism'." So he opened the play with two motives: woman and socialism. He wanted to be a famous socialist and a world-reformer like Babel, with whom he identified himself in the dream; also he wanted to conquer women. He had failed in both respects. It will soon be seen that the unconscious confused these concepts. He did not want to get, but he wanted to be, a woman; and he did not want to be a socialist, but to conquer a certain socialist—to have him as a life companion.

When he was 14, his friendship with a youth named Constantine began. They read together French socialistic literature. Constantine went to a revolutionary center at Petersburg. Fedja did not go because of fear of his father. Constantine became a hero, was soon disappointed in Fedja and became more and more cool to him. Sexually, Constantine—a normal lad—was interested in girls and saw little of Fedja. He was a good speaker, writer and organizer. During the World War he went to the front, whereas Fedja, at that time a state official, remained at home. Constantine lived now in Paris and was known there as one of the most famous of the émigrés. In trying to match Constantine's great talent and energy, Fedja over-reached himself. Shortly before Fedja's illness began, Constantine married in Paris.

Dream 2: "I was walking with my friend Constantine. He was smoking a cigaret. I was astonished because I knew that he did not smoke. As he had nearly finished his cigaret, I offered him another. He gave a few puffs, then threw it away, saying 'This cigaret is no good'."

Fedja offered himself in the belief that Constantine might already be tired of his wife, but Constantine could not do anything with Fedja.

Behind the friend, Constantine, one sees the shadow of a certain Dimitri, another friend and school companion, who later became an actor. At about the same time that Fedja became friendly with Constantine, this Dimitri attained considerable corrupt influence over Fedja. He taught him masturbation and caused Fedja frequently to have uncanny emotions. Fedja frequently decided never to see Dimitri again, but he felt an irresistible attraction to him. Even at the time of presentation, Fedja felt goose-flesh when he remembered this hypnotic dependence of that time. He then suffered from headaches, and asked his father to put him into another school, but this was not done. Once he got up at 1 a. m. and went to the bathroom, because he thought it was morning and time already to get up. Here one sees a far advanced sign of the later disease. This sex dependence lasted for several years until Dimitri had no more interest in Fedja. He also began normal sex life. Fedja went once or twice to prostitutes, and allowed them only to touch him. He found them insufficient substitutes for the demonic Dimitri. Never in his life did he have any other sex experience after the loss of Dimitri. He frequently dreamed of Dimitri.

Dream 3: "I wanted to go to bed. In my bed was a man—an actor. I took off my clothes and took my night-shirt out of the bed."

Dream 4: "I lay on a bench. An old school companion lay down beside me. He put his arm around me. I said he should not do it because . . . . then he retired."

The sublimation of Dimitri into the admired Constantine was plain enough. The thread which led backward from Dimitri to the patient's brother, Alexis, was not so clear. In their youth, Alexis teased Fedja constantly until the latter did or said something which put him in the wrong. Alexis was glad when Fedja broke out in temper. At that time, he was subject to temper tantrums, whereas at the time of the analysis he seemed to be quite empty of any emotion. Once Fedja threw a knife at Alexis without hurting him. Alexis used this impulsive act as a reason for not speaking to Fedja for two years. Both boys lived at home as enemies.

The two brothers represent the Russian soul and its contradictions—both shipwrecked; Alexis became a saint; Fedja escaped into disease and dreamed

much of his brother. Always when he spoke of his brother he became restless, sometimes yawned, sometimes even had seizures.

The homosexual component of Fedja, discovered in analysis, could be isolated on the basis of the transference to the analyst. The specific character of the illness made the analyst able to breed the homosexual component as in a bacillic culture. As it is not the aim of this presentation to give the whole case which ended with a remarkable result, I have to add to this history only the experiments which prove that the seizures of Fedja contained distinct homosexual fantasies.

Dream 5: "I ordered coal, but it was too expensive. I asked, 'Will it not become cheaper'?"

I explained to the patient that the coal was energy—perhaps sex energy which he lacked—but what he had to pay for it—his special complexes—were dear to him. He would like to retain them and still recover, and therefore he asked if this would not become cheaper. During this explanation, I saw the first seizure of petit mal: slow chewing commenced, tetany of the left hand, staring for two minutes; he uttered the words "Yes....Now....What is it?"

In the next session, the patient announced a desire for a physical examination of his heart, and he demurred at going to another physician when I explained that I was not willing to examine him.

The second seizure in my office took place while the patient was reporting that he had gone to a physician for the heart examination. After several dreams in which he described clearly enough his homosexual tendencies toward his brother, we discussed homosexuality in general. Fedja said that homosexuality had always been incomprehensible to him. He always avoided the word "homosexuality" and said "this" or "that" instead of using the word. I asked him if he meant that homosexuality was disgusting. As if at a cue, the patient's face became blank; then there appeared an expression of nausea and salivation at the mouth. The left hand was in his pocket. He arose from the chair and took off his coat. He then sat down and took off his shoes. I was afraid of a more severe attack and ordered the patient to lie down on the couch. He said "Yes," but continued to undress. He looked at his watch and after five minutes awakened. The patient saw his shoes by the chair, was embarrassed, and said "I must have had a spell." Never before had he undressed during his attacks. The transference to the analyst became more and more clear. He identified me with Dimitri, Constantine, Alexis and his father.

Dream 6: "I was dictating to Dr. Wittels out of my manuscript, 'Handbook of the Russian State Rights.' I told him that he might type it. Dr. Wittels replied that it was all the same to him. He could just as well write it by hand."

In this short dream the word "hand" appears three times: namely, "manuscript," "handbook" and "write by hand." The patient had dictated to his sister before her marriage. She was his secretary. It was the same to him whether it was the sister on her machine, or I with my hand; i. e., whether hetero or homo.

One day, the patient's father remarked that it was a long time since Constantine had written. The patient replied by having a spell. Shortly after, there was a rumor that Constantine was coming to Vienna. Fedja had three spells, one after the other, and the fourth one this day in my office. We discussed at that time the subject of sexual intercourse and the fact that many persons considered everything sexual to be vulgar. The patient said that this was the opinion of theosophists, for example, of his brother, but he had another view of it. This time I tried to arouse an attack experimentally, using the word which I had learned to be a magic influence; I said, "But we have to admit that there are perverted forms of sex life which are really *disgusting*." Again, as at a cue, the patient lost consciousness; he salivated, exhibited spasm of the hand and this time undressed until he was really quite naked. First, he took off his coat and trousers, laid them, well folded, on the couch, then took off his collar and his necktie. During his undressing, I called his name loudly. The patient said, "Yes,.... *But I must undress first.*" The patient then proceeded to dress again, but he was not quite clearly oriented, and so the session ended.

Two days later, the patient came again, but without a dream to report. This was for Fedja an exceptional thing and showed resistance. I decided that the time had arrived to explain to him his homosexual tendency and the transference to the analyst. He could see that he was playing a woman's rôle—that he wished to share life with Constantine, the beloved friend, whom he identified with Dimitri, with Alexis and with me. I explained to him that he needed the unconscious for a fantasy, which was unbearable and impossible for his ego. According to his physical constitution, he used attacks for experiencing his fantasies. At the end of the session, Fedja stood opposite me and asked me some trifling question. In this moment, he had another seizure. His hat fell out of his hand, he scratched his head and smelled his fingers; a blissful smile appeared on his face (anal component). He sat down, recovered consciousness and went home.

No more spells occurred in my office until much later. One day, after the analysis, he returned to pay me a visit. I remarked that I did not consider this analysis efficient, especially in the direction of his brother. At this moment, again the expression on his face changed, the chewing movements of his jaw began and his right hand had a cramp. He got up, took off his coat and vest and laid them, folded, on the couch. I shouted "Wake up, Fedja." He looked at me slyly and put his finger on his mouth, as if to say "Be silent." Then he sat down as if highly satisfied. Four minutes later, he dressed, following my direction, and was soon himself again.

I could describe two more cases in which I could experimentally produce a seizure in epileptic patients and observe, just as distinctly as in Fedja's case, the psychic content of it. I wonder whether these experiments, exposing the repressed sex fantasies of epileptic patients, have ever been published! As I could discover nothing of this kind in the literature, I am inclined to attribute to myself the discovery of this possibility. I repeat that I do not consider the production of the seizures themselves as the essentially new point in my contribution. This has been described before. But it is new to follow the patient into the seizure itself and to reveal a psychic content in a spell, which certainly has an organic basis, and was considered, not so long ago, as being only organic and empty of any psychic mechanism.

DISCUSSION

DR. L. PIERCE CLARK: I think that I may best discuss this paper of Dr. Wittels by detailing briefly my own experience in this field.

A number of years ago, I made a composite picture of the character make-up of the essentially epileptic person. The data used were carefully gleaned from the literature plus my own detailed observations and study on public and private material of many years' standing. As a result, the essentials of such a character were shown ideally in egocentricity, supersensitiveness and emotional poverty. In the main, most epileptologists have accepted this descriptive picture of the epileptic subject. However, if one allows this syndrome to remain at a descriptive level, the psychology of the epileptic patient will be little better than a mere detailment of the microscopic observations in the epileptic brain which Meyer has aptly termed the symptomatic pathology of the disease. My best efforts were foiled in trying by ordinary clinical methods to go further in the process of refinement of the syndrome by separating it into its component parts. I next studied the fragments of mental content of epileptic patients during and following certain types of seizures when ordinary consciousness was disturbed or actually lost in automatic deliria. I found that the mental content at such periods was surcharged with other egoistic strivings, and especially with sadomasochism of varying intensity. This conclusion, as well as the character, have been known for many years. My clinical researches only confirm and further refine the known psychology of the epileptic patient. Then, some ten years ago, I tried to employ freudian psychoanalysis, disregarding the symptomatic picture of the fit and even the obvious character fault. But ordinary analysis was not possible in the absence of a transference as in the psychoneurotic, so I employed largely the narcissistic transference similar to that employed by those who attempt psycho-

analysis in the psychoses. As a result of these labors, I am prepared to state, tentatively, that the epileptic form of narcissism, which name is now used to replace the older form of classic descriptive syndrome, is capable of showing the well known reaction formations of sadomasochism; secondly, there is found hardly any fear formation. In point of fact, as is also well known, the essentially epileptic person is almost fearless of his disease as well as of any outer foe. Indeed, he is the bravest of the brave, for he instantly represses his fears and only occasionally experiences them at the aura of a heralded attack. The mystery why the epileptic subject does not normally permit himself to experience fear or its congeners of anxiety and panic holds the reason why he is not neurotic in the phobic sense. Behind this would-be fear formation lies the essential extreme dependency of the infantile neurosis. Is this infantile dependency of identification with the mother only different quantitatively from that seen in the ordinary neuroses? I infer that it may be, as it is slowly and incompletely displaced in the analytic transference, the latter being purely narcissistic in type. But this may be stressed too prematurely, as in many neurotic persons the final character analysis shows similar forms of narcissistic or pseudonarcissistic elements which are not dissimilar to those seen in many epileptic subjects. In many cases, the narcissistic formation is so protective of the infantility of the ego that it may easily lend the impression that its origin is organic or at least constitutional. While this statement may seemingly give comfort to a histologic pathogenetic theory, I would warn that many cases that are seemingly organic have turned out illusory, as the complete relief in epilepsy of long standing has shown, even though so-called organic dementia was in evidence. I believe I have shown that the vast majority of essentially epileptic persons possess a species of narcissism which on precise analysis reveals sadomasochism, and that the deepest root of this defect is a dependency or identification with the organic mother.

Is there a specific type of narcissism for the epileptic character as distinct from other clinical pictures of narcissism? I do not know and am not prepared to outline it more precisely at this time. In many persons with epilepsy, one finds a pattern that is closely interwoven with the ego structure, independent of the narcissistic element; it impresses the fact that at least one is dealing with some psychic defect in the unconscious that is a deep defect in the organismic pattern which defies present psychoanalytic ability to penetrate. I have found, however, that with patience one may go deeper and deeper. I have also found that though the epileptic narcissism is seemingly a pure culture of this defect it always possesses a variable element of objective transference, and it may be it is this saving element that renders analysis more successful the more it is present. Did one go no further than what I have briefly sketched here, the gain in knowledge of the psychology of the epileptic patient would be enriched, but still not operable as a theory or hypothesis. I will therefore undertake to state in broadest outline a psychoanalytic formulation of the disease in the reverse order of our analysis, by stating that the epileptic make-up is not so dissimilar to a supernarcissistic character neurosis; that its primary defect lies in the infantile neurosis of extreme dependency or identification with the organic mother; that the reaction formation of an extremely repressive force prevents fear reaction as a defense, and hence the person with epilepsy is not neurotic nor does he react off his "fear of fear" that continually impounds his libido in the unconscious, where it has a tendency to return into consciousness as a sadomasochistic or fit-reaction. The fit is then the libidinal counterpart of the repressed libido that is not reacted off by egoistic activity nor objectivated in normal healthful living and sufficient libidinal satisfactions.

DR. A. KARDINER: I should like to express a word of appreciation of Dr. Oberndorf's paper before proceeding to that of Dr. Wittels. It contained an unusual wealth of material and raised many interesting points which it is, however, too late to discuss.

Dr. Wittels' paper I found agreeable. There is only one issue on which I wish to comment. Dr. Wittels evidently analyzed a man who happened to be

epileptic. There is nothing to object to in that. Epileptic subjects have complexes just as all other people have. Though Dr. Wittels did not expressly say so, he intimated that the complexes he described provoked the epileptic reaction. This inference I do not believe is justified, nor does anything he said prove the point. It is one thing to say that in an epileptic seizure the patient vents some homosexual trends; it is another thing to say that in order to express his homosexuality he gets a seizure. The latter is extremely unlikely. Dr. Wittels would have us believe, as he put it in his introductory remarks, that the epileptic person is one who has a tendency to fits. If one were to apply this type of reasoning to anxiety hysteria one would say that a person with anxiety hysteria is one who has a tendency to anxiety; and if you were to ask, "What have the complexes to do with the anxiety?" you would have to infer that the complexes merely touch off the anxiety. That is not, however, what psychoanalysis teaches. Psychoanalysis has conclusively demonstrated that the anxiety is the very meat and fabric of the complexes, not merely a reaction type. As regards the epileptic reaction, no one has hitherto shown the path of regression which produces the epileptic reaction. For these reasons, I maintain that the approach to epilepsy from the point of view of the neuroses and schizophrenia is sterile and productive of nothing but arbitrary assertions. Although Kraepelin does not enjoy the following he had forty years ago, the fundamental principle on which he worked remains unchallenged by any psychopathology that has followed. His fundamental premise was that a difference in symptomatology warrants the assumption of a difference in pathology. On this account, I maintain that the transplantation of the pathology of schizophrenia to epilepsy is a serious error. I may mention in passing that I have worked on the subject of epilepsy from an entirely different approach — from the reactions produced by trauma — and find the existence of factors that pass one by if epilepsy is regarded as a phenomenon produced by libido regressions. From this approach, one begins to get some idea of the nature of the epileptic seizure itself, and not merely as Dr. Wittels and Dr. Clark have shown, the uses for which the seizure is used. As a pathologist one can readily agree that it is put to some pleasurable use. But that is not a great discovery and moreover should not detain one from looking beyond it. Psychoanalytic theory as it exists today does not supply sufficient material to elucidate the nature of the epileptic seizure, and that is where the essential problem lies.

DR. SMITH ELY JELLIFFE: The fifth edition of the textbook by Dr. White and myself came into the office this morning. In lugging out this new child for your admiration, I would ask you to note that the energic point of view concerning the epileptic attack is there elaborated in such a way that it seems to me it is stupid to talk about a pure fictional antithesis, organic or functional. This is to me nonsense. No matter what the person has, he still has fundamental strivings to go after a special goal. This is his entelechy or pattern. He has to go somewhere, and no matter what the character of the machinery may be, he is going in that direction. The nature of the machinery will alter the mode of his strivings, but whether it happens that in the machine there is interposed a tumor of the brain, an encephalitic infiltration, a syphilitic inflammation, a gliosis or I do not care what, nevertheless, the goal is always to be thought of. It seems to me that Dr. Wittels should say that he gave us a paper on the psychology of *an* epileptic patient. There is no such thing as *the* epileptic patient. There are a lot of *an* epileptic subjects. The epileptic patient is a monstrous abstraction. In discussing the psychology of an epileptic character, one finds innumerable degrees of involvement of the mechanisms which Dr. Wittels has so beautifully set forth.

I want to say a few things about what I said a moment ago concerning the wisdom of the Greek poets. If one reads of the mad Hercules of Sophocles, it will be noted that he kills his two sons; then one reads Hippocrates' celebrated treatise on epilepsy. These illustrate the fact that the dramatist knew more about the human drama and what was going on in the soul of the individual than the scientists, even as great as Hippocrates was.

There are two small points that I would bring up. Dr. Wittels said that in his patient's first dream there "was a sort of Babel." I wish to give an empiric association with Babel that might add to Dr. Wittels' interpretation. The Holy Rollers are a sect that roll around the floor in convulsive distortions, and strive for the capacity to talk all languages that were talked in the mythologic Tower of Babel originally. So far as language is an expression of our actions, originally hand actions, therefore, Babel's many tongues equal many hands, many arms, many phalli. The patient would wish to possess innumerable phalli; therefore, we get a glimpse of this omnipotent wish and, vice versa, of his impotency.

About the coal: In coal there is a symbol of anal eroticism. It seems to me that these two component parts of the dream, the polyphallic fantasy on the one hand, and the anal eroticism on the other, with the scratching and smelling one sees in the monkey house, are confirmatory evidence of the patient's intense sadism which he must check, and can do so only by becoming absolutely impotent, i. e., by becoming unconscious. The epileptic attack represents a lust murder, or, rather, a running away from lust murder, if you will, expressed in every different patient with differing degrees of intensity. The destruction, or murder, being symbolized behind an innumerable series of signs. Sometimes, the patient does not run away from the object of his sadistic impulse successfully, as when in an attack he attacks himself, for one finds violent lust murders occurring in the epileptic attack.

One thing else — Dr. Wittels spoke of the conscious capacity to induce a specific attack as a new contribution. I am not certain that is quite so. Most of the members of this society have heard many of us speak of complex indicators, and the principle of conditioned reflexes is too well known for one not to realize that this is what is here involved. Some of you will remember a patient with selective rumination whom I described before this society. She could, at will, bring up into her mouth strings of celery or seeds of strawberries or other objects eaten at a meal. After I had known her for a long time, I knew just what kind of criticism to direct to the patient in order to have her bring up one of the objects eaten. If I always agreed with her sarcasms, ironies or even witty sadisms regarding other men or women about her all was well — there was no rumination. But as I practiced mild or more marked demurrals to her criticisms I could play on the stomach's contents almost as if it were a bargain counter and cause her to bring up any desired food object swallowed.

DR. WITTELS: It is always a great pleasure to hear Dr. Jelliffe talk and I must thank the American men who have made it possible to talk in this society about the psychology of epilepsy. You are much more advanced than the neurologic societies of my country. There it is nearly impossible to talk about what American neurologists call organic diseases in connection with psychology and psychoanalysis. When I tried to get some patients from a superintendent of a certain hospital in order to examine them, he said: "Am I to send an epileptic to an analyst? It is meaningless." It is not so obvious to every one as it seems to be here that epilepsy has a psychologic content. It is true that one can incite a good many narcolepsies by inciting their complex. My contribution was to show that this can be done in epilepsy. With them not only a seizure breaks out, but it is highly interesting to me that behind this loss of consciousness there is something in the inside of the patient, and I can say that it is a reexperience of a scene that he had experienced when he was a child; so I should like to close my speech before this society with a note of appreciation of the men who have made it possible to talk on this subject: Dr. Jelliffe, Dr. William White and Dr. Clark.

Book Reviews

HANDBOOK OF INDIVIDUAL PSYCHOLOGY. Edited by DR. ERWIN WEXBERG, Vienna. Price, unbound, 46.50 marks; bound, 89.80 marks. Vol. I, pp. 664; vol. II, pp. 200. Munich: J. F. Bergmann, 1926.

This exhaustive treatise succeeds in presenting the essentials of individual psychology up to the time of its publication. Since the appearance of Alfred Adler's original study of organ inferiority, in 1907, a literature has grown up about this fruitful conception which demonstrates again the widespread interest which the newer psychology in its various phases has excited, extending far beyond the field of medicine as ordinarily conceived. The first impression of Wexberg's monumental work is its scope. It is probable that no such comprehensive attempt to elucidate the problems of life, as applied both to the individual and to society at large, has ever been attempted on the basis of a single theory. This universality of application, which finds full expression in these volumes, may well lead to a decided modification of the basic thesis.

Adler himself does not appear as a contributor except for a brief but important preface. In these preliminary remarks Adler states in no uncertain terms what he considers the aims of individual psychology, and one stands a little appalled at its comprehensiveness. He believes that in the study of internal organs has been found a biologic and anatomic-pathologic foundation which brings many questions of a biologic sort nearer to solution. The body-mind problem has found a presentation which throws new light on the innermost relationship of the integrity of organs and the mental life. In fact, the new doctrine may be expected to help toward a solution of the problem of life in all its manifold relationships. Therefore, the task, he thinks, is set to build up individual psychology as a philosophy of life. At the same time the demand is insistent that the problem of the neuroses, difficult children, and crime as manifested in the individual may be attacked by methods which the new point of view has revealed. Above all, prophylaxis is the essential aim. Individual psychology takes its place among the great movements which seek through their universal application to remove the difficulties which stand in the way of progress.

This ambitious program depends for its realization on the study of the individual, and through him of the social conditions of which he forms an integral part. The individual psychology is far from being an abstraction of metaphysical character, but demands above all a technic and training in an appreciation of the problems of the individual in their most practical aspects. The aims and accomplishment of this important phase of psychologic theory and practice are set forth in exhaustive detail in nearly 1,000 pages of closely written type in which upward of thirty contributors participate, among them a goodly number of women, the whole being put out under the names of Alfred Adler, Bruno Krause, Eleonore Rieniets, Karl Sulzer, Max Fürnrohr, Fritz Künkel, Leonhard Seif, Verploegh-Chassé and Egon Weigl.

The table of contents gives an idea of the scope of the two volumes. In the first, as a somewhat general introduction, individual psychology as science is discussed by Kronfeld of Berlin, followed by chapters on organ inferiority, on method and sources of cognition and on the "Begabungs" problem. A second section is concerned with child psychology and pedagogics under many subheadings, in which are discussed such matters as the mental development of the child, the nervous and difficult child, his inner life, his school life and his relation to the family and finally his relation to the courts. A third section of the first volume is of more clinical import. Under the general heading, psychopathology, the following topics are discussed by as many different writers: The pathologic structure of the neuroses, neurasthenia and hysteria, compulsion neuroses, disturbances of speech, sexual aberrations, schizophrenia in the light of individual psychology, manic-depressive insanity and the technic of individual psychologic treatment. All of these chapters have a direct bearing on the practical application

of the doctrine as applied in medical practice. The dictum that pure psychoneuroses are not diseases, but rather mental states which may be psychologically understood and pedagocically treated is elaborated in detail and with much cogency, by Wexberg. Seif contributes a chapter on the compulsive neuroses to which is appended a detailed report of a number of actual cases, a method of presentation adopted also by several others.

A second volume, a third the size of the first, develops the broader aspects of the application of individual psychology to philosophic questions, sociology and criminology. Religion, ethics, politics and a variety of allied subjects in their relation to the new approach are discussed at the hands of eight writers. A bibliography of 337 titles, followed by a name and subject index closes this notable contribution to psychologic point of view, which has gained many adherents through its human appeal and noncontroversial presentation.

Whether it will ultimately be possible for the inferiority idea, as developed by Adler, to bear the heavy load which its followers are placing on it remains to be determined. That the idea and its implications are and will be most fruitful as a means of approach to fundamental questions relating to the psychoneuroses and to the inner life in general is no longer to be questioned. The temptation to draw deductions from a single principle is difficult to resist, and it is probable that the adherents of this relatively new psychologic point of view will find it difficult to maintain the wide generalizations of this first formulation. There can be no doubt, however, that such a presentation as Wexberg has given will secure many followers who have found the freudian hypothesis unsatisfying in its broader social aspects. As a continuation and, in a sense, elaboration of Freud's fundamental concept, without which it could hardly have come into being, the individual psychology will take its place as a stimulating contribution to a psychologic movement, which, however, must be considered still in its infancy. It is regrettable, for the benefit of the ordinary reader, that the volumes might not have been condensed. Had this been possible it is safe to say they would have had more readers and the gospel thereby have been more widely diffused.

REPORT OF AN INQUIRY INTO THE AFTER-HISTORIES OF PERSONS ATTACKED BY ENCEPHALITIS LETHARGICA. By ALLAN C. PARSONS. Reports on Public Health and Medical Subjects No. 49. Price, 4 shillings and 6 pence. London: His Majesty's Stat. Off., 1928.

This is a study of the late results in encephalitis, based on vital statistics, hospital records, reports of local health boards and units of experimental encephalitis in England from 1919 to 1927. It supplements two reports previously published.

There have been approximately 16,000 cases notified, with 7,500 deaths, but the author believes that this represents only from 60 to 75 per cent of the actual number of cases. The monograph, with a large number of tables, indicates on a large scale the public health aspect of the disease and its epidemiologic character to some extent, but lays chief emphasis on the fate of persons who have been attacked by the disease. It would appear from the gross figures that "if one hundred cases are investigated, say, three years after the primary illness, the average findings will be as follows: patients who have survived without serious consequences, 25; patients who have died, 35; patients who have become more or less disabled, in mind or body, or both, 40."

A long catalog of symptoms is given, but it is manifestly incomplete. Conduct changes in children are considered in some detail with the efforts made along various lines to correct and treat these disorders. The author has studied various civic and social service institutions which are involved in the care of these troublesome and incorrigible children, and, although no conclusions are drawn, one is enabled to appreciate the extraordinary difficulties that surround the proper handling of these patients. He shows that these patients do not fit in anywhere in the scheme of present-day treatment of delinquency, and, except for some special encephalitis units in connection with hospitals or reformatories, he sees only the

adaptation of selected prisons for such cases. An appendix with over 300 case histories is attached. A prefatory note by George Newman says: ". . . the incidence of the disease, as measured by the yearly notifications, is small when compared with that of most other communicable diseases, and that in terms of population, the total amount of disability and fatality caused by the disease is not so great. In terms of cases however the matter is much more serious . . ."

THE CHILD AND SOCIETY: AN INTRODUCTION TO THE SOCIAL PSYCHOLOGY OF THE CHILD. By PHYLLIS BLANCHARD, Ph.D., New York: Longmans, Green & Company, 1928.

At present there seems to be a paucity of literature by which a beginner can orient himself in the field of social psychiatry—that which is available either being intended for popular consumption or being too advanced to be of value without some preliminary knowledge of the subject. It is this gap which this monograph seems excellently qualified to fill. Although social psychiatry is such a young science that many of its conclusions must be tentative, there is a rapidly growing body of material that seems to indicate that the adult personality results from the interplay between the person's innate equipment and the social setting of his childhood, and that the latter factors, because of their variability, exert a more powerful ultimate effect than the former. Furthermore, because these factors are capable of alleviation, they can be modified in order to aid the person's adjustment. The author has surveyed these factors as they exist in present civilization and discusses their effect on the child, basing her conclusions on numerous case histories drawn from a varied personal experience with the problems of children. Starting with a review of the ways in which emotional responses may be modified, she indicates how this modification of response underlies the manner by which the child becomes a socialized individual. In this process, the family, the school and the modern social environment, including the significance of such forms of modern recreation as the moving picture theater, each play a rôle. Their effect and also the effect of the child's intelligence, his play, religion and reading are discussed in separate chapters of part I.

In part II, the reasons for failures in socialization are discussed on the basis of the author's experience with children, and are illustrated by case reports.

At the end of each chapter topics for discussion are listed. There is a glossary of difficult terms and an adequate bibliography. The book can be recommended to physicians—particularly pediatricians, students of psychiatry, psychologists and teachers.

ENCÉPHALITE EPIDÉMIQUE. By DR. RENE CRUCHET. Price, 15 francs. Pp. 136, with 8 illustrations. Paris: Gaston Doin, 1928.

In the preface by Achard, an attempt is made to claim for Cruchet priority in the reporting of encephalitis. He states that in medicine the discovery of a microbe gets for its discoverer more notoriety than does the report of a new disease, and to Achard this seems an injustice. This book is an effort to lay claim to this priority and to record the sixty-four cases seen by the author since his war service from September, 1915, to date; on the cover the author states "The 64 first known observations." The first case described was that of a soldier who was seen by the author on Sept. 15, 1915, and the second in December, 1915.

The second chapter is devoted to the cases that are labeled encephalomyelitis that were seen at Bar-le-Duc in 1916 and 1917. The cases are divided into the following: (1) mental forms; (2) cases with symptoms of meningo-encephalitis; (3) convulsive forms; (4) choreic forms; (5) hemiplegic types; (6) cerebellar and pontocerebellar types; (7) bulbar types; (8) medullary types; (9) polyneuritic types. The entire volume is written in the case history style, with only a rare pathologic report.

This little paper-bound volume is an addition to the already voluminous literature on encephalitis, but is valuable in that the earliest cases are reported and an insight is given as to their type. Any one interested in the clinical aspect of this disease will do well to read over at least one or two of the type cases.

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